JUNE 1, 1949

MODERN MEDICINE

The Journal of Diagnosis and Treatment

SYMPOSIUM ON DIABETES

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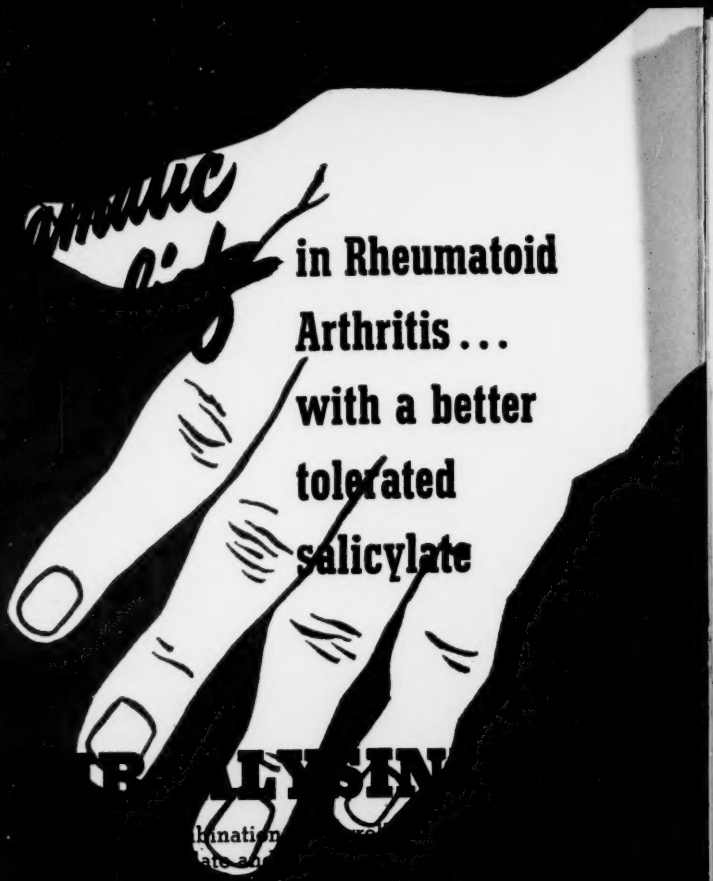
Dr. Charles H. Best *(see page 44)*



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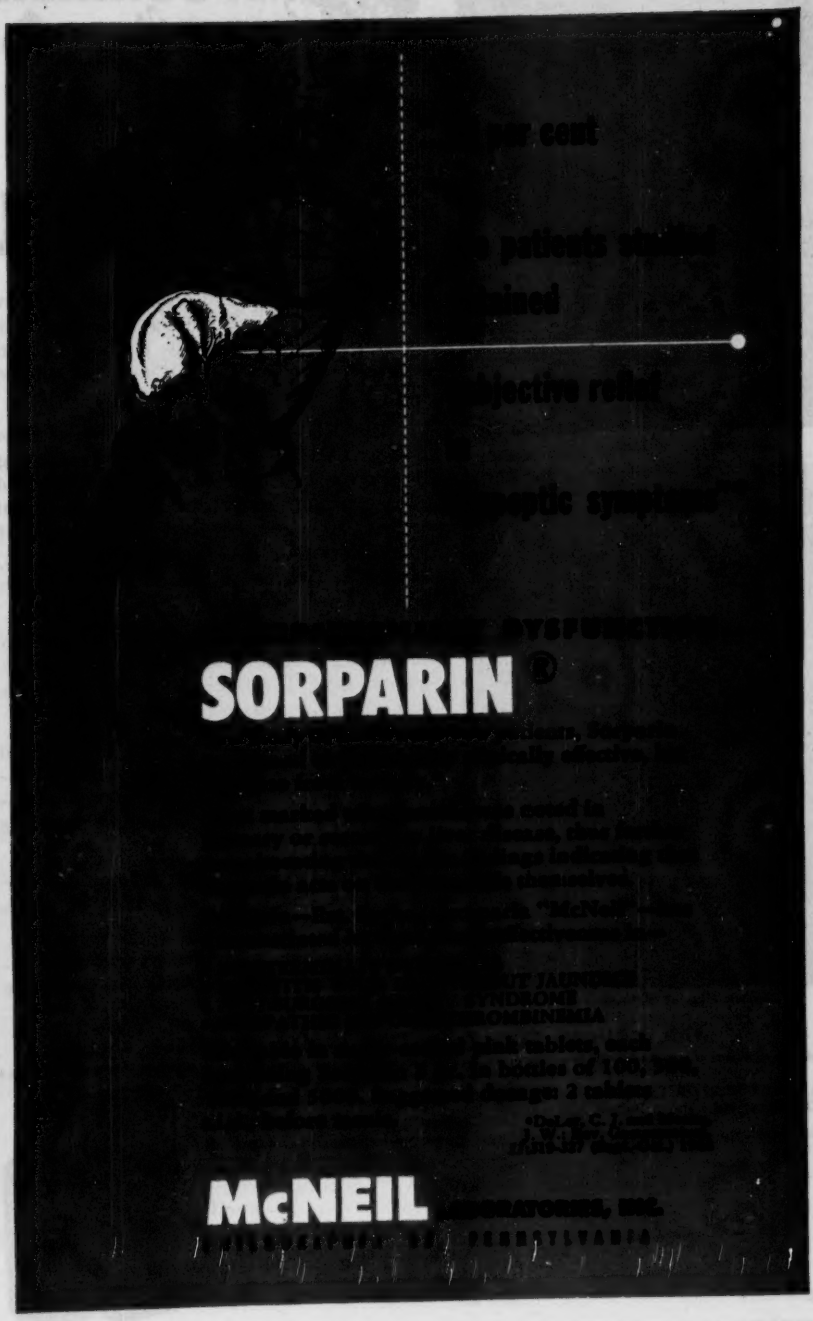
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LETTER FROM THE EDITOR

Dear Reader:

The Symposium on Diabetes, starting on page 43, speaks for itself. But the fame of this special issue had spread even before the first pages came off the press.

Letters started coming in about the symposium while we were still struggling with the galley proofs. In the middle of April we were pleased to read the following note from Dr. Hugh L. C. Wilkerson, Diabetes Section of the U.S. Public Health Service, Boston:

Several months ago I heard of your plans for this special issue from a friend of mine in Washington, D.C., whom you wrote regarding suggestions, I believe. It is pleasing to me to learn that this diabetes issue is now scheduled for a definite time.

We have four demonstration units geographically distributed throughout our country. Indeed we are interested in every aspect of diabetes mellitus and try to make available in our library all information we can gather on the subject. Would it be possible for you to send us five extra copies of this special issue for permanent placement in the library at headquarters in Boston and in the libraries of each of our field units?

We were particularly tickled with a note from one of the contributors to the symposium, the Grand Old Man of Diabetes, Dr. Joslin. We share it with you. Dr. Joslin wrote:

You may know that I am to go to Brussels, June 2, and talk before various medical societies in Belgium, Switzerland, and possibly elsewhere. If you have proofs of the papers which are coming out in Modern Medicine *in June could I see them, because they would help me to be up to date.*

After you have perused this issue, we hope that you, too, can say that you are up to date with Dr. Joslin.

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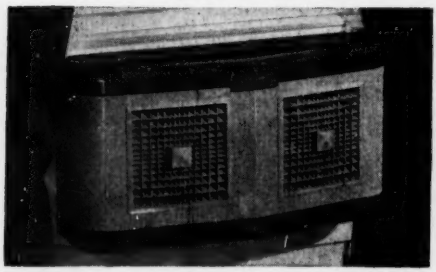

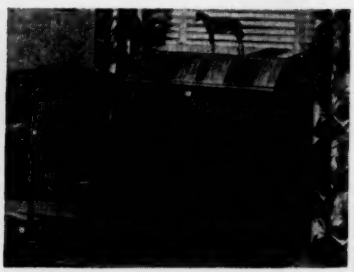

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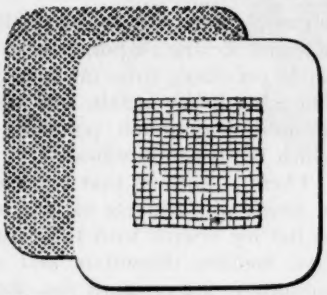

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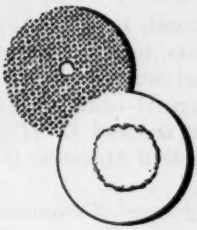

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Correspondence

Communications from the readers of MODERN MEDICINE *are always welcome. Address communications to The Editors of* MODERN MEDICINE, *84 South 10 St., Minneapolis 3, Minn.*

Penicillin for External Otitis

TO THE EDITORS: I would like to add an observation to the very comprehensive report dealing with the treatment of external otitis by Jack R. Anderson, M.D., and C. H. Steele, M.D., in *Modern Medicine* (Mar. 1, 1949. p. 75).

I treated 2 cases of extensive fungoidal external otitis of both ears which had been treated for over one year with different remedies, including thymol and furacin, as follows:

After cleaning with hydrogen peroxide, the ear canal was dried and packed with 50,000 units of pulverized penicillin. After twenty-four hours the ear canal was almost free. I repeated the treatment once. Since then the patient has been cured.

ADOLF REICH, M.D.

New York City

Case for Bal Understated

TO THE EDITORS: In a recent issue of *Modern Medicine* in a discussion of the treatment for poisoning by various substances, there appears the statement that Bal *may* be used in therapy for arsenic poisoning (Mar. 1. 1949, p. 36). I believe that this constitutes an understatement and should read, "Bal must be used."

I think that the experience of many has now placed Bal as the treatment of choice for arsenic poisoning as well as for poisoning from mercury, gold, and other heavy metals, with the exception of cadmium poisoning, for which Bal is contraindicated.

There is nothing that I am aware of which will compete as successfully as Bal for arsenic with tissue sulfhydryl. Sodium thiosulfate and other monothiols are certainly less efficient.

All provisions in emergency rooms and elsewhere for treating acute poisonings should include a supply of a 10% solution of Bal in peanut oil to which about 20% benzyl benzoate has been added.

FRED G. HIRSCH, M.D.

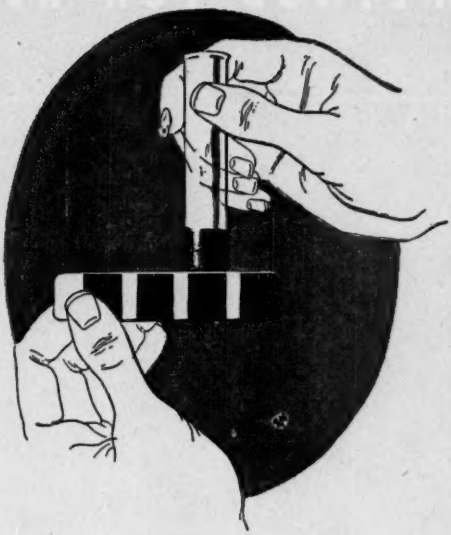
Army Chemical Center, Md.

Differ on Diagnosis

TO THE EDITORS: I would like to comment on the answer to the question on episodes of apparent transitory paralysis of the left arm in a nineteen-month-old male infant (Dec. 1, 1948, p. 24) and on a criticism of this answer in correspondence (Mar. 1, 1949, p. 20).

May I offer in diagnosis the condition known as subluxation of the head of the radius. The age of the child, the history, the position of the apparent-

(Continued on page 18)



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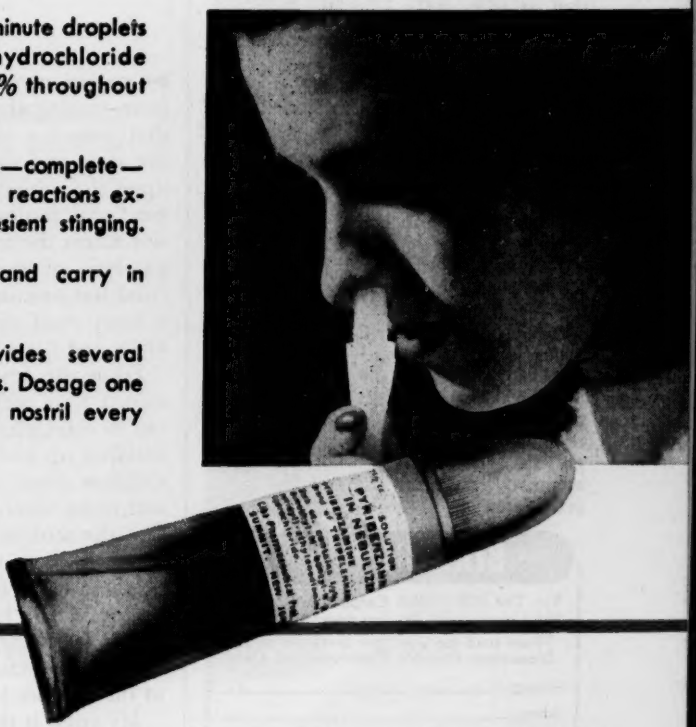

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ly paralyzed arm, the pain on motion, the lack of deformity, the recovery during the next twenty-four hours without treatment, and the recurrence of the condition all strongly favor this diagnosis. This may be confirmed as the treatment is instituted by placing the thumb over the radial head and applying pressure while the forearm is being supinated. A characteristic click will be felt at the radial head as it is reduced.

In the past year I have seen 2 such cases. I refer to Boyette and London, *Journal of Pediatrics* 32:278, 1948, for a complete summary of this subject.

HAROLD S. COLEMAN, M.D.
Los Angeles, Calif.

► TO THE EDITORS: Usually I refrain from writing the editor, feeling sure that someone else will probably do the same and that light will be shed upon the subject without my wasting time and stationery. But the discussion anent the cause of the temporary paralysis of the nineteen-month-old child has seemingly degenerated into a hunt amid the volumes in the library and the musty tomes of history.

From the first appearance of this case, I have seen vividly the numerous mothers who yank their stumbling children up and down the curbs, the children often dangling by one arm and often taken off guard so quickly that the muscles are undoubtedly relaxed. I have wondered why more of these cases have not reached the doctor or the hospital. Perhaps they do in later life. Perhaps many and interesting causes are then ascribed to the injuries found.

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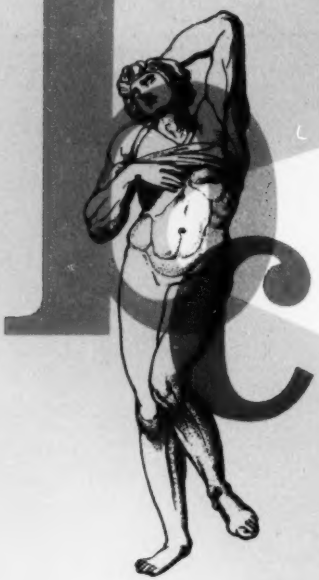

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the child very much, and is generally irritable and impatient. Also that the child is overweight, poor muscled, and living on a high-carbohydrate, low-protein, low-vitamin diet. Who wants to bet?

ELIZABETH L. BROWN, M.D.
Avon-by-the-Sea, N. J.

Every Doctor Should Read

TO THE EDITORS: For the good of all living and future doctors, send reprints of Case MM-139 about the man with a glioblastoma to all editors and publishers of medical literature *(Modern Medicine,* Mar. 15, 1949, p. 86).

It is interesting, it is picturesque, it is alive. It is the best medical reporting I have ever read.

H. L. PUCKETT, M.D.
Stillwater, Okla.

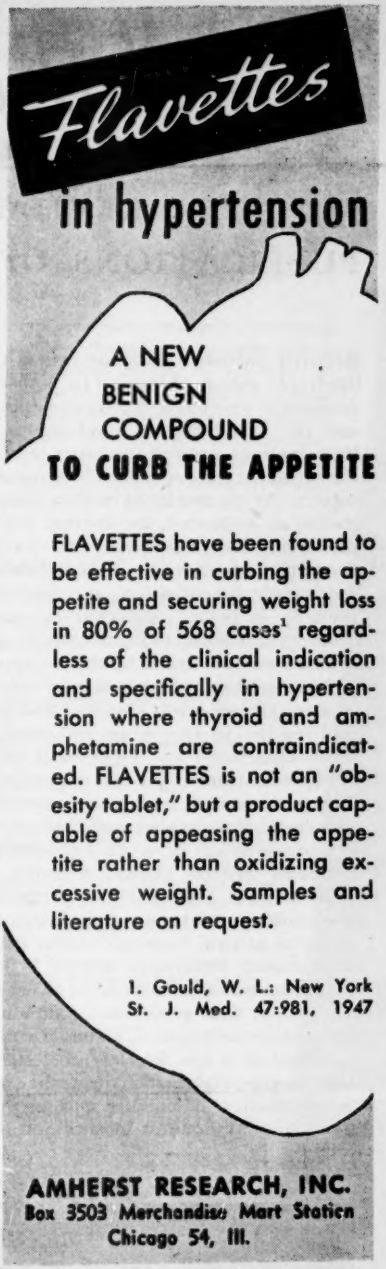
► TO THE EDITORS: *Modern Medicine* is read and studied so eagerly by so many physicians that I think an important omission in Diagnostix, Case MM-139, should be corrected. In diagnosis of the brain tumor, no mention of examination of the eyegrounds was made—an oversight, I suppose. By the way, I should like to know how many G.P.'s are equipped and able to do ophthalmoscopy, also how many are equipped to do and do microscopic urinalysis in their offices.

H. W. LIEPMANN, M.D.
Central Valley, N.Y.

Best, Bar None

TO THE EDITORS: I would like you to know that I consider *Modern Medicine* the best magazine on medical topics that I receive, bar none.

MORRIS FRANKLYN H. LEVY, M.D.
Brooklyn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A REVIEW OF RECENT PUBLICATIONS OF CLINICAL INTEREST

I

Recently published data concerning the lipotropic action of inositol in both experimental animals and human subjects, and the broad clinical implications of these findings, prompt this series to keep the physician informed on this important subject. As the results of further clinical studies are published, the findings will be presented. This member of the vitamin B complex appears to possess significant therapeutic potentialities, and intensive research is being conducted in many quarters to establish indications for its use.

In a study of various lipotropic agents administered to 111 patients with atheromatous disease it was observed that inositol resulted in an average reduction of blood cholesterol and cholesterol esters of 19 per cent and 17 per cent respectively. The patients with hypercholesterolemia manifested this drop in blood cholesterols in a period of 6 to 10 weeks. The same individuals showed clinical evidence of atheromatous coronary artery disease. The author points out that lipotropic agents in general "warrant further study as promising therapeutic agents."[1]

Inositol was administered to a 37 year old patient with generalized xanthomatous cutaneous lesions. Collateral therapy consisted of a low fat, low cholesterol, high protein, high carbohydrate diet, and administration of choline and methionine. Inositol alone or inositol and diet markedly reduced total blood lipids and also phospholipids, but cholesterol esters and total cholesterol remained high. Despite these objective changes and symptomatic improvement of the patient the author warns that lipotropic activity can be properly evaluated only after prolonged study.[2]

Goldstein and Rosahn have successfully employed inositol combined with choline in the treatment of patients suffering from cirrhosis of the liver in an advanced stage.[3] More recently, Broun[4] reported complete recovery of a patient with advanced hepatic cirrhosis, who received daily doses of 1.0 Gm. inositol, 1 Gm. choline, and 2 tablets of fortified yeast. Previous attempts to control ascites with choline and liver extract therapy had failed. The dietary regimen was kept unchanged.

INOSITOL-C.S.C.

Inositol-C.S.C. is supplied in 0.5 Gm. capsules in bottles of 100.

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". . . an improved sense of well-being, increased appetite, a more normal mental state, more restful sleep, less pain and, in almost every case the patient becomes very much more optimistic."[2]

tolerance to ERTRON

The use of Ertron in rheumatoid arthritis "has been characterized by almost complete absence of toxic effects, despite serum calcium concentrations sustained at high concentrations . . ."[3]

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Questions & Answers

All questions received will be answered by letter directed to the petitioner; questions chosen for publication will appear with the physician's name deleted. Address all inquiries to the Editorial Department, MODERN MEDICINE, *84 South Tenth Street, Minneapolis 3, Minnesota.*

QUESTION: I have 2 patients who at periodic intervals develop severe aphthous stomatitis. What therapy would you recommend for this condition?

M.D., New Jersey

ANSWER: *By Consultant in Dermatology.* Unfortunately the cause for this condition is not yet known with certainty. Some observers think that specific allergic reactions to food may produce this eruption in some cases. Certainly this is not the common explanation.

Probably the condition is a manifestation of infection with the virus of herpes simplex. If this is true, it is reasonable to try to prevent the condition by giving repeated vaccinations with cowpox vaccine as for prevention of herpes simplex in other areas. Details of the technic are open to differences of opinion, but a series of 6 or 8 vaccinations given at two-week intervals is probably advisable.

QUESTION: What is the most effective treatment for a rash due to use of hair dye? The rash appears mainly on the face and neck of the patient.

M.D., Indiana

ANSWER: *By Consultant in Dermatology.* In dermatologic practice the specific causative agent seldom determines a specific choice of therapy. In this case the nature of the cause should be disregarded and the morphologic features of the eruption treated. As no description of the eruption is given, I can only suggest the following:

For a mild dermatitis, a carbolized lotion may be sufficient. If the eruption is subacute, zinc oxide paste, possibly with the addition of ichthyol, naphthalan, or crude coal tar may be employed. For a more acute eruption with moisture, oozing, or vesicles, cold compresses must be used. Ultraviolet radiation is often helpful for subacute eruptions, and roentgen radiation for chronic cases.

QUESTION: A sixty-year-old patient who had had angina pectoris and slight dyspnea on exertion for ten years asked me to give him injections of male hormone, from which he claimed great benefit was derived previously. He had moderate hypertrophy of the left ventricle; blood pressure was 130/95. Three weeks ago I started 25-mg. injections of testosterone every two days. After the second injection the patient stated he was feeling well. After the fourth, he developed left ventricular failure. This condition has improved with bed rest, digitalis, salt-free diet, and so forth. I feel that testosterone was the precipitating factor. What is your opinion?

M.D., New York

ANSWER: *By Consultant in Cardiology.* Testosterone therapy may cause sodium and chloride retention. It is reasonable to assume, therefore, that acute cardiac failure may result from excessive dosage.

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WARREN-TEED

Forensic Medicine

COMPILED BY ARTHUR L. H. STREET, LL.B.

PROBLEM: May a medical expert witness base an opinion as to a patient's physical condition upon the symptoms related by the patient?

COURT'S ANSWER: No.

The Texas Court of Civil Appeals, Amarillo, applied the general rule that a doctor can testify to an opinion based on x-ray study and objective symptoms disclosed by an examination of the patient, but not upon what the patient has told him. However, when a doctor testified on direct examination that an opinion was partly based on what the patient had said, the defect in his testimony was cured by his statement on redirect examination that he would have reached the same conclusion upon the examination he made of the patient and an x-ray study (214 S.W. 2d 873).

PROBLEM: Could a patient's right to recover damages from a food dealer, on a theory of food poisoning, be supported by the doctor's testimony that when he treated the patient, seven years before the trial, he was of the opinion that illness was caused by food poisoning?

COURT'S ANSWER: No.

The Ohio Court of Appeals, Summit County, said that the doctor could have testified to facts learned by him in attending the patient and that he could express, as of the time he was called as a witness, an opinion as to what caused the illness, but that the case came within the general rule of law that "a medical expert should state the opinion he holds at the time he testifies, rather than the opinion held at some previous time" (83 N.E. 2d 657).

The court cited decisions of appellate courts of Illinois, Vermont, and Massachusetts to the same effect.

PROBLEM: Does the fact that a druggist refuses to fill a physician's prescription so reflect upon the physician as to entitle him to damages from the druggist?

COURT'S ANSWER: No.

In so deciding, the Louisiana Supreme Court noted that a druggist may have the best reasons for refusing to fill a prescription. He may not have the necessary ingredients or, as a chemist, he may perceive that the physician erred in the formula. A druggist may distrust his own ability to prepare the prescription, or other causes may disincline him from undertaking to fill a prescription.

However, in this case the court adjudged the druggist liable for slander in having publicly stated that he would not give a straw for the particular doctor's diploma and that the doctor had issued one prescription that would have killed the child for whom it was intended (La. Sup. Ct 26 L. R. A. 325).

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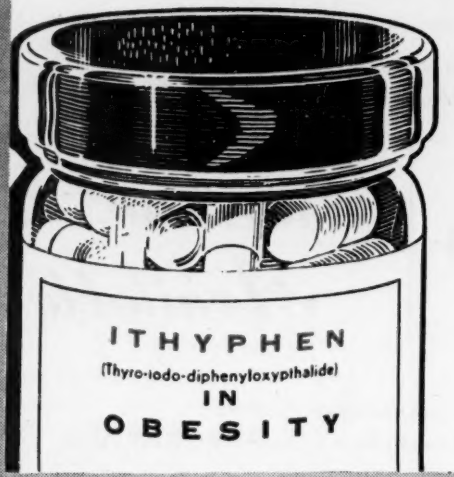

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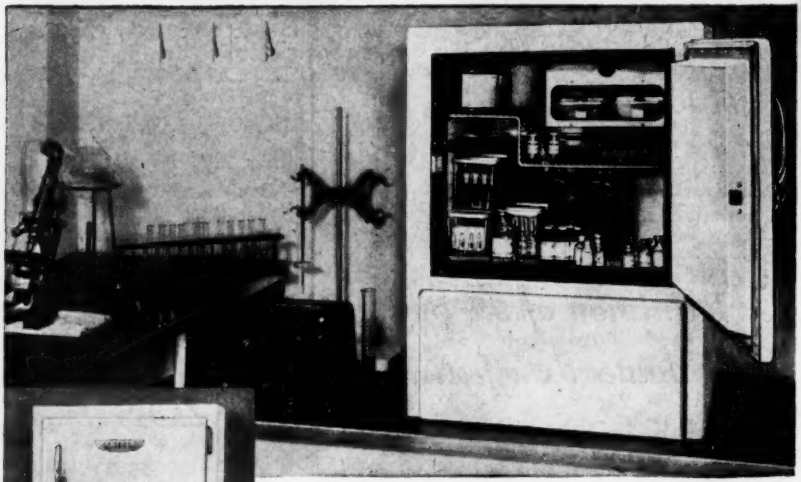

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Washington Letter

No Health Insurance Bill This Session

National compulsory health insurance probably won't be taken seriously by congressional leaders before the next session. Two developments in the last weeks offer no encouragement for President Truman's insurance plan:

1] The committee which will handle the Truman bill is planning to hold hearings between the end of this session, late in July, and the opening of the next in January.

2] Sen. Elbert Thomas (Democrat of Utah), chairman of the Senate Welfare and Labor Committee, has announced that the bill hasn't much chance this session.

Committee Split Also Dooms Bill

A deep split within Sen. Thomas' committee also is adding to the difficulties of President Truman and his Security Administrator, Oscar Ewing. Sen. James Murray, Montana Democrat, who heads the subcommittee considering the Truman plan was not given jurisdiction over *all* health legislation.

Another subcommittee headed by Sen. Lister Hill, Alabama Democrat, is handling all other "local aid" bills. Thus every national health bill except the Truman proposal has been turned over to Sen. Hill.

Hill, though a liberal, has refused to support the compulsory insurance plan. In fact, he is the chief sponsor of a separate bipartisan health bill which resembles Sen. Taft's bill in most particulars but spells out in more detail the status of a medically indigent family. The bipartisan plan would specifically eliminate the means test and would allow each state to define a medically indigent family.

"Friends and relatives nothing. Just people trying to get a doctor!"

(Continued on page 34)

The pioneer contributions by the eminent Thomas Addison in his study of the endocrines was a significant phase in the development of the field of Endocrinology. The Armour Laboratories, as a pioneer in this field, is keenly appreciative of Dr. Addison's early studies and illuminating descriptions.

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Thomas Addison, M.D. 1793-1860

Thomas Addison's major contributions to medicine are bywords to every physician and student of medicine—but he, himself, is little known. Even in his lifetime, Addison kept much to himself and was apparently wholly absorbed in the study, practice, and teaching of medicine. He was born at Long Benton, near Newcastle, England, in April 1793. He received his M.D. degree from Edinburgh in 1815 and began practice in London. He became expert in skin diseases by working under the distinguished dermatologist, Dr. Bateman, at the Public Dispensary. In 1824 he was appointed assistant physician at Guy's Hospital, and in 1837 was made full physician and joint lecturer on medicine with the illustrious Dr. Bright. Addison published an essay "On the Operation of Poisonous Agents," and also a number of shorter medical articles, each of which displayed remarkable diagnostic acumen and attention to detail. He made extensive studies on pernicious anemia which he termed "idiopathic anemia" and which, to this day, is often referred to as "Addisonian anemia." At first he apparently confused this condition with "disease of the suprarenal capsules," which he described and which was subsequently named "Addison's Disease" by Trousseau.

Committee Favors Noncompulsory Plan

The situation has slipped so far out of administration control that a majority of the Thomas' committee is committed *against* the Truman plan. A favorable committee report is unlikely but might result if a tremendous popular demand develops or if the President puts full pressure on the senators.

As to the first contingency, what sampling has been done indicates that the general public's attitude varies between confusion and ignorance of the facts. The second contingency, too, is highly unlikely despite the President's message to Congress. Even Vice-President Barkley is believed not prepared to demand passage of a compulsory health insurance act at this session.

However, there may be plenty of fireworks over this issue even before Congress adjourns.

A Surprise from Britain

A Physicians Forum meeting in Washington received some unexpected advice from a member of the Royal College of Physicians. Dr. Thomas M. Galloway of Edinburgh praised the operations of the national health plan in Britain, but said that the United States isn't prepared for such a program. He pointed out that Britain has been working up to such a system for forty years. The Physicians Forum has endorsed President Truman's plan.

Why We Get Cold Remains a Mystery

After two years of experiments in Fairbanks, Alaska, the Aero-Medical Laboratory researchers admit they don't know why one man gets cold and another doesn't. They have turned up some miscellaneous data about the north country, some of which already is proving valuable, but they admit they're still puzzled as to why 2 men of the same physical type, wearing similar clothing, react differently to the same frigid temperature.

The group is under direction of Maj. Gen. Malcolm C. Grow, the Air Surgeon. Some of their positive findings are:

Don't rub a frozen face with snow or take a drink of whisky to keep warm. Instead, use warm water for the application and forget the whisky; alcohol dilates facial pores and quickens the cooling-off process.

Geographic origin doesn't seem to make much difference in reactions to cold, and excessive fat is no protection.

You don't have to eat regularly to survive or operate at 35° below zero. One test group had nothing but water for six days and at the end the men said that they felt weak but could have gone on longer if necessary.

Science Foundation Bill Snagged

For the third time in as many congressional sessions, legislation to create a national science foundation has run into trouble over administration. Sen. Thomas' bill now is in a favored position, but opposition is growing.

The main objection is that this bill leans too heavily on committee work and does not provide for enough executive power. In these respects it is an improvement over the bill that President Truman vetoed two years ago.

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Attention has recently been drawn to "an interesting approach"[1] to the control of psoriasis and neurodermatitis. Perlman[2] has reported that following the oral use of undecylenic acid, psoriasis was relieved completely in 3 out of 17 patients, and was partially relieved in the remainder. "Relief of itching is sometimes noticed as early as two days after institution of treatment . . . undecylenic acid seems to hold a great deal of promise in the improvement and possible prevention of recurrences of psoriasis and neurodermatitis."[2]

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1. Undecylenic Acid and Psoriasis, editorial, J.A.M.A. 139:660, 1949.
2. Perlman, H. H.: J.A.M.A. 139:444, 1949.

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Case MM-144

THE CLUE

ATTENDING M.D: The patient sitting in the wheel chair is sixty-six years old. His chief complaints are dyspnea, aching around the heart, and chronic cough. Three years ago he began to feel weak and run-down. He soon had difficulty walking because, as he says, his legs would give out. He was treated with digitalis by his physician. Two years ago, while turning, he suddenly felt dizzy and had the sensation of choking. He fell into a chair, uttered a few gurgling sounds, and lost consciousness for a minute or two.

VISITING M.D: Did anyone notice convulsive movements?

ATTENDING M.D: His sister was there and noted some twitching of the eyelids, nothing else. He had never had any convulsive seizures. The next day he noted palpitation and exertional dyspnea and remained in bed for several weeks on his doctor's advice. Last year he suffered two similar syncopal attacks.

VISITING M.D: Has he ever been here before this admission?

ATTENDING M.D: I saw him in the outpatient dispensary two months ago and noted severe peripheral arteriosclerosis, cardiac enlargement, a blowing systolic apical murmur, and apparently heart block. The ECG revealed 2 to 1 block with varying conduction time. Neurologic examination was normal.

VISITING M.D: What led to his coming to the hospital this time?

PART II

ATTENDING M.D: While driving his car a week ago he felt as if he were going to have another attack of unconsciousness. Despite his staying at home after that, mostly in bed, the exertional dyspnea increased, he was dizzy, and had pain over the eyes and palpitation. He decided to enter the hospital.

VISITING M.D: Please tell me all the pertinent past history.

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Aspergum simplifies post-tonsillectomy care—shortens convalescence.

Children welcome this form of medication—they like the flavor and chewing gum form of Aspergum. Each tablet of Dillard's Aspergum contains 3½ grains of aspirin. White Laboratories, Inc.. Pharmaceutical Manufacturers, Newark 7, N. J.

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White's *Vita* **A D** *&* **Ointment**

for faster healing

Supplied in 1.5 oz. tubes, 8 oz. and 16 oz. jars and 5 lb. containers.

ATTENDING M.D: The usual childhood diseases. During adolescence he had a febrile illness with hemoptysis and loss of weight. He denies venereal disease, and has not had rheumatic fever. During the past ten years he has gradually become shorter. His height was formerly 5 ft. 10 in., and is now only 5 ft. 7 in.

VISITING M.D: That is interesting, but probably unrelated to the present illness. Wassermann? X-ray films of the bones?

ATTENDING M.D: Serologic reactions for syphilis were negative on two occasions. *(Handing him films)* The x-ray pictures of the femurs and tibias show changes of osteitis deformans, so-called Paget's disease. Calcification of the arteries is also visible. The right clavicle has a distinctly fuzzy appearance, like loose cotton wool. The skull appears normal. Nevertheless, Paget's disease may be limited to one bone. Kyphosis and calcification of costal cartilage with widening of the lower thoracic aperture are common, with a "violin"-shaped trunk.

VISITING M.D: Since this seems to be an incidental finding, we shall proceed with the physical examination. Temperature is normal. *(Examines patient in bed)* Percussion is impaired over both lung apexes. Apex of heart is in fifth interspace to left of median line where a harsh murmur is heard, transmitted to the axilla, and a fairly loud second sound. These sounds correspond to radial pulsations, with long intervals between impulses. No diastolic murmur. Blood pressure 180/70. Large pulse pressure, common in heart block. What about other laboratory work, including the patient's chest films?

ammonia dermatitis

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Boston, Massachusetts

*Address by R. H. Aldrich, M. D.

PART III

ATTENDING M.D: Heart is enlarged, and old fibroid tuberculosis scars are in both apexes. Red blood count 6,000,000; hemoglobin 110%. White blood count 5,000, normal differential. Urine normal. Sedimentation rate 20 mm. in one hour; sputum contained no tubercle bacilli on three occasions. Pulse rate varies between 35 and 45. ECG yesterday, ventricular rate 35, atrial 84. Regular rhythm, no conspicuous relationship between any two P and R waves. T_I and T_{II} are negative.

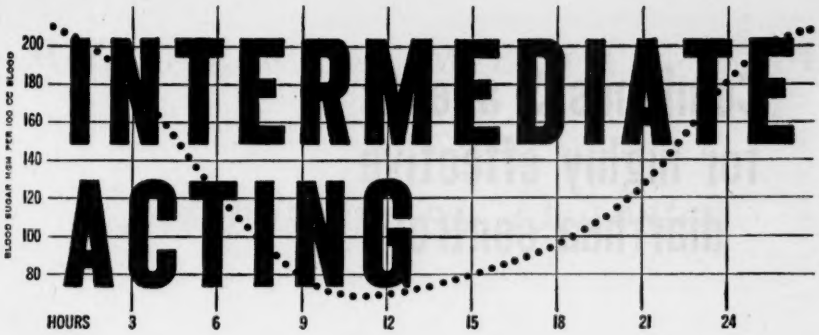
VISITING M.D: Complete heart block exists. There appears to be an atrial arrhythmia on this ECG. This condition often occurs with heart block. You will find an excellent description of ECG features of heart block in Thomas Lewis' book *The Mechanism and Graphic Registration of the Heart-Beat*. Cause of this heart block?

PART IV

VISITING M.D: The possibility of syphilis has been eliminated. Block due to digitalis usually does not persist and, although prolonged digitalis therapy can cause heart block, this case is probably an example of Stokes-Adams syndrome.

ATTENDING M.D: Arteriosclerosis?

VISITING M.D: Yes. The signs of myocardial insufficiency, the dilated heart with a relative mitral insufficiency, and the other features make me think that the atrioventricular bundle of His is damaged. There is always the possibility of chronic myocarditis, on a rheumatic basis. Most cases with heart block show extensive myocardial damage and not an isolated lesion in the bundle.



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1. Rohr, J.H., and Colwell, A.R.: Arch. Int. Med. 82:54, 1948.
2. ibid Proc. Am. Diabetic Assn. 8:37, 1948.

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Symposium on Diabetes

Foreword

CHARLES H. BEST, M.D.*
President, American Diabetes Association

DIABETES continues to occupy the foremost position among clinical conditions the study of which provides new findings to illuminate all aspects of medicine. Its position is not even seriously challenged and this is understandable when we define it as a disturbance of carbohydrate, fat, and protein metabolism due to an imbalance of the glands of internal secretion.

Experimentally, all permanent diabetes is characterized by absence or deficiency of normal beta cells of the Islands of Langerhans. The burden of proof is on the clinical pathologist who states that the beta cells function normally in any case of diabetes mellitus. He will not be able to *prove* his contention at present, but new facts may, of course, come to his rescue. In all probability the patient required insulin and the means were not at hand to determine whether there was a deficient supply or an abnormal demand.

A great deal of evidence indicating a deficit of insulin is available in many cases, but we await a satisfactory method of determining insulin in blood before we can seriously embark on the search for the diabetic who has his disease in spite of a normal endogenous supply of the hormone. In the early stages of diabetes produced by anterior pituitary extracts, or by the adrenocorticotropic hormone, there may well be a good supply of insulin for a short time. In the completely depancreatized

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animal, which of course has no insulin, the severity of the diabetes may be increased for a time by administering diabetogenic materials. It is quite possible therefore to imagine a situation in which the beta cells continue to function during the diabetic state, but many of us are persuaded by the evidence available that their struggle to survive is likely to be as short as it is vigorous.

Our generation of research workers will leave plenty of problems for the next. An advance like the discovery of insulin answered a question—there is an internal secretion of the pancreas! We do know more about its action than about that of any other protein hormone. These studies of the action of insulin have taught us a vast amount of physiology and biochemistry, but the final goal is not yet won. When it is, I can visualize the flood of fascinating new problems revealed by observations from the new vantage point.

None of us would wish all the physiologic problems to be solved—our minds might then atrophy like unused muscles. But we do hope, someday, to make the "ideal" insulin, which will be liberated only in response to need, so that the diabetic will never have hypoglycemia or hyperglycemia. We do aspire to prevent *all* the complications of diabetes, to eliminate any obstacles which make it difficult for the insulin-treated individual to make the same contributions to society which the insulin-secreting person can make.

THE MAN ON THE COVER is Dr. Charles H. Best who is also author of the Foreword to the Symposium on Diabetes featured in this issue of *Modern Medicine*. The man in the background is Sir Frederick G. Banting. Dr. Best began research on the internal secretion of the pancreas with Dr. Banting in 1921 in the laboratory of Professor J. J. R. Macleod at the University of Toronto. From their work came the discovery of insulin. In 1923 the Nobel prize in physiology and medicine was awarded Dr. Banting, who shared it with Dr. Best, and to Professor Macleod who shared it with Dr. J. B. Collip. Dr. Banting was killed in an airplane accident while on a mission to England in World War II. Dr. Best is director of the Banting-Best Department of Medical Research and head of the Department of Physiology, University of Toronto.

The Future Diabetic

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Prepared for Modern Medicine

EVERY time since 1898 that I have summarized my statistics on diabetics, the death rate has fallen and the length of life increased. The duration of the diabetic life has risen from 4.9 to 14.1 years, and average age at death has advanced from 44.5 years to 64.5 years.

Will this trend reverse itself in the future? I cannot believe it.

It is obvious, however, that the lengthening duration of the disease and the older and older age at death cannot proceed at so rapid a rate. Two-thirds of all our cases begin after the age of forty years and three-quarters of the living cases are now over forty. Since the ordinary expectancy of life for us all at that age is only a trifle beyond thirty years, each new patient with diabetes who is over the age of sixty-one years must exceed the normal life expectancy for his age or else he injures my statistics!

We have traced all our 476 diabetic physicians treated in the last half century. The results with this group as a whole show what can happen when knowledge of the disease has been acquired and is applied. The death rate of those doctors who were between twenty-five and thirty-nine years of age at onset of disease is only half that of all diabetics of similar age. The results are not so striking at other ages. However, only 2 doctors have died of diabetic coma since 1931.

Two other groups of statistics encourage us to look forward with confidence to the future of diabetics. In Dr. H. C. Shepardson's group, which consists of 50 of our patients with onset under forty years of age, 34 have survived the disease for twenty-seven years and only 2 of these are incapacitated.

The third group consists of 31 children with onset in the first decade of life. They lived with diabetes for 19.1 years.

In the final analysis, the future of the diabetic must and will continue to be largely in his own hands. It can be bright, if he will control his disease. Today the means are available.

THE NEW MILLION

What a splendid opportunity for improving the treatment of diabetes is offered to young doctors today! At their door, ready to be diagnosed and treated, are the million as yet undiscovered diabetics.

The outlook for these future patients is far brighter than for our present known million. The unknown patients are asymptomatic, unhandi-

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capped by other disease, with a short duration since onset, and only mild involvement, if we dare to call any diabetes mild. They are largely the relatives of our present known million.

These patients will know more about their disease from the start, for they can't avoid hearing about it.

There will be a need for many more diabetes camps, thousands of teaching and nursing homes, and special provisions for the old. I believe so fully in the unusual abilities of diabetic patients that I wish they could provide for themselves and for research in diabetes. But this will not be the case. They will not be left alone. The other 146,000,000 people of the country will gradually learn that these patients are test objects, 2,000,000 human guinea pigs, and that if their premature aging is postponed, the whole world will profit.

Meanwhile the course of diabetes and of diabetics in the future, as in the past, will depend upon alleviation of the present causes of death and upon better treatment of the disease itself.

CAUSES OF DEATH

What are the causes of death of diabetics today? Why do they die?

Tuberculosis—Only a half century ago, and still more recently in cities like Vienna and Paris, probably half of all diabetics succumbed to tuberculosis. It was far and away the chief complication Bouchardat (1806-86) encountered.

Today in our diabetes cases, death from tuberculosis has fallen to only 2.5%.

According to figures of the Metropolitan Life Insurance Company, tuberculosis is now almost eliminated. Diabetes as a cause of death has passed tuberculosis and pneumonia, so greatly has the conquest of these diseases rather than our clinical efforts aided the diabetics.

Pneumonia—In my early days it was said that all diabetics acquiring pneumonia died. I disproved that many years ago.

Even two years ago pneumonia and other general and local infections caused only 7.8% of our total deaths. And under infections we included not only systemic but local infections, appendicitis, gallstones, abscesses, and acute renal infections.

Diabetic coma—Probably almost as strange a cause of death in Bouchardat's time as today was diabetic coma. Oddly enough, it came to the front coincidentally with the discovery of its cause—the overfeeding of fat—in the period 1898 to 1914. At least 14 of Naunyn's 49 autopsied cases died of it; of von Noorden's cases 64% succumbed to it, and of ours, 62%.

As soon as F. M. Allen opened our eyes to Bouchardat's thesis not to overfeed a diabetic, our percentage fell to 40% and, in our recent 2,225 deaths, to 1.7%.

These last 35 coma deaths which occurred in various parts of the country among my former diabetics, I shall publish, because they represent the underprivileged, ignorant, uncooperative, and carelessly treated cases. They drive home how infinitesimal in importance is any discussion of the minutiae of the treatment of diabetic coma with or without alkalis or with or without glucose, compared to its prevention.

Cancer—The deaths from cancer have risen from 1.5% between 1898 and 1914 to 8.9% between 1944 and 1946. This is quite natural with the age at death advancing from forty-four to sixty-four years and the duration for exposure of the individual being nearly trebled.

Will the percentage continue to rise? Diabetics must die of something. They are not immortal. I hope the rate will not go up materially and I wish it might fall.

The diabetics should solve the question of whether health examinations are worth while. What other group is more assiduously followed up? Doctors are on the spot when it comes to cancer in their diabetic patients. The doctor who treats diabetics should be a well-rounded physician and not a diabetes specialist.

Arteriosclerosis—At the commencement of the century, 17% of my diabetic patients died of arteriosclerosis in its various manifestations, today 67%. Is the condition really hopeless? Take gangrene, one of its worst and best-known forms. Our mortality has dropped from 8.1% a little over a decade ago to 2.9%.

But, as for arteriosclerosis in the heart, the kidneys, and the vessels generally, is there any hope? I think so.

In the first place, we definitely believe that the arteriosclerosis of children with diabetes of long duration is similar to that of adults. Here is the precise statement upon which our group agrees, and this also reflects the ideas of Dr. Shields Warren and his staff:

> In the most recent autopsies upon diabetic patients of 25 to 39 years of age who have had diabetes for periods varying from 15 to 30 years, examination of the arterial system by pathologic techniques shows advanced changes which in morphology do not differ from those seen in advanced arteriosclerosis of non-diabetics of more advanced age. Renal arteriolar sclerosis is marked and in the larger arteries, intimal atheromatosis and calcification in the media are commonly present. It is the much greater frequency of advanced arteriosclerosis and particularly its malignant character in young diabetic patients of long duration which distinguish it.

Convinced as we are about the essential similarity of arteriosclerosis in the diabetic and nondiabetic, may I direct your attention to certain facts learned in our clinic about diabetic coma:

Years ago Root and Bloor found that 1 in 6 of our recovered coma cases developed tuberculosis within the subsequent five years. Still more recently Dr. Priscilla White has observed that among our children with diabetes of twenty or more years' duration, 15% who showed no arteriosclerosis had not had coma; the 40% who had obvious arteriosclerosis had had coma one or more times, and the 60% who were incapacitated by their arteriosclerosis had had coma repeatedly.

No one ascribes to coma alone the cause of arteriosclerosis, but coma represents a state of uncontrolled diabetes. We say to a boy today, "If you wish a successful career, control your diabetes." To a girl, "If you want a baby, control your diabetes." Intermittently but steadily, we are developing support for this advice.

The premature arteriosclerosis in diabetics appears in unexpected regions and conditions. Dr. Priscilla White has called attention to the calcified pelvic arteries of a pregnant diabetic girl and the prognostic value

for the fetus—1 chance in 10 for a living child without exceptional care, and only 1 chance in 5 with the best.

MEDALS

Years ago we gave medals to our patients who lived longer with their diabetes than their nondiabetic neighbors of equal age were expected to live. Over 2,000 of our patients have earned such recognition. An analysis of these should be valuable, because they have overcome premature old age.

But we are far more concerned with a higher medal, far more difficult to obtain. This is our so-called Quarter Century Victory Medal, which is awarded to those rare persons who after twenty-five years of diabetes have bodies unscathed by the disease, with no vascular complications detectable by a clinician, an ophthalmologist, and a roentgenologist.

When I ran across the first case, what a thrill I experienced. To see that healthy man of thirty-nine years with twenty-five years of diabetes behind—the first medalist—was like being at Kitty Hawk and watching the Wright Brothers' airplane rise, or reading of Blériot when he made the first channel flight from France to England, or of Lindbergh when he flew the Atlantic. There is always a first. Now there are 10 of these medalists.

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Current Research Problems in Diabetes

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Prepared for Modern Medicine

BOTH for the researcher in his animal laboratory and for the clinical investigator, the abnormalities of metabolism presented by the diabetic patient have constituted a continuing source of fruitful investigation over a period of many years. Our present knowledge of metabolic processes and, in fact, the current birth of scientific endocrinology stem from basic facts obtained during early investigations of the diabetic state.

In a short review of research problems in diabetes one is faced with the decision of either listing many or discussing a few. The author has chosen the latter approach, with full realization that problems not mentioned may prove of far greater importance than those discussed.

VASCULAR COMPLICATIONS

Probably the most urgent and practical problem presented for investigation by our enormous diabetic population concerns the vascular complications or vascular associations of the diabetic state. From the work of many investigators[1] comes the irrefutable conclusion that existence of diabetes for a period of fifteen to twenty years is associated with a very high incidence of vascular disease—vascular nephritis, hypertension, vascular retinal degeneration, and coronary, cerebral, and peripheral vascular disease.

These vascular manifestations, which affect arteries of all sizes, arterioles, capillaries, and veins, are related to the duration of the disease more closely than to the age of the patient. Use of insulin over the past twenty-eight years has not only been ineffectual in preventing these lethal changes but has actually brought the problem into the limelight by making it possible for diabetic patients to survive long enough to manifest them.

If inadequate control of the diabetic state during all or part of the patient's diabetic life is responsible for the degenerative vascular changes, such changes are properly labeled vascular complications of uncontrolled diabetes. We would then have the right to hope that, with newer preparations of insulin, offering better physiologic control than can be attained with the tools now at our disposal, these complications may be preventable.

If on the other hand these vascular manifestations should prove to be independent of the defects in the metabolism of carbohydrate, protein, and fat observed in the uncontrolled diabetic but, nevertheless, phenomena associated with and comprising

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an integral part of the entity called diabetes mellitus, it would be erroneous to regard the vascular lesions as complications. The approach to prevention would then mean prevention of diabetes itself, with little hope of influencing the incidence of vascular lesions in the patient who has just developed the disease.

The answers to these questions are not yet available. Extensive studies are being carried on to elucidate this vital problem. Until clear-cut evidence can be marshaled to prove either concept, it would seem wise for physicians to direct therapy toward the goal of metabolic normalcy, often unattainable at present, rather than to throw caution to the wind, allowing grossly abnormal metabolism to exist and using insulin only to prevent ketosis and to allow survival.

INSULIN PREPARATIONS

The problem of producing the ideal insulin preparation has a number of interesting facets for the investigator. A nonprotein substitute which could be taken by mouth has not yet appeared upon the horizon and may be many years away.

However, a step in this direction has been made. Recent discoveries indicating that hormones influence the activities of specific enzyme systems may point the way.[2, 3] As our knowledge increases regarding the various sites of activity of insulin, it is conceivable that a compound unrelated chemically to insulin will be discovered with similar qualitative influences upon the same enzyme systems.

Assuming that no such substance will be found in the foreseeable future, the next best approach would be the preparation of a depot type of insulin geared to release its activity in relation to rises and falls of blood sugar level.[4] This difficult problem is receiving thoughtful attention in several laboratories.

In the meanwhile, the various mixtures and modifications of insulin now at our disposal represent definite advantages to many patients. However, we are keenly aware that many diabetics cannot be adequately controlled, in the physiologic sense, with available tools.

The insulin problem cannot be concluded without mention of the so-called alpha cell hormone. It has been realized for years that most preparations of insulin, even the highly purified ones, contain a substance which elevates the blood sugar, and that this activity is soon masked or overcome by the hypoglycemic activity of the preparation.

Recent investigations[5] indicate:

1] The blood sugar–elevating activity of this substance is exerted via increased hepatic glycogenolysis.

2] The substance is protein in nature and is precipitated along with insulin by the methods used commercially in this country.

3] Destruction of the hypoglycemic property of the insulin proper with the use of cysteine unmasks the blood sugar–raising principle, the latter being unaffected by cysteine.

4] After destruction in animals of the insulin-producing beta cells of the pancreas by means of alloxan, the concentration of the blood sugar–raising principle in the pancreas follows the distribution of the remaining islets of Langerhans in a remarkable relationship. Since alpha cells comprise the

bulk of surviving islet tissue after alloxan destruction of beta cells, it has been suggested that the blood sugar–raising principle is an alpha cell hormone.

This important observation could explain why the alloxanized diabetic animal with pancreas in situ requires more insulin for control than when his pancreas has been removed. In addition, it brings up for revaluation and reorientation a number of important clinical problems that have hitherto defied rational explanation.

ETIOLOGY OF DIABETES

The relentless increase in our diabetic population serves to emphasize again the need for more and more information relating to the factors which *initiate* the cycle of events that eventually produce the permanent diabetic state. Any approach to the prevention of the disease must await disclosure of these factors and of the mechanisms through which they operate. It seems to be true that a hereditary factor exists and that obesity frequently pulls the trigger for the production of clinical manifestations[5] in those who carry this trait.[6] When one has made this statement he has covered essentially all that is known clinically about the etiology of the garden variety of diabetes mellitus.

By the time the patient shows the diagnostic criteria of the existence of the diabetic state, he is presenting, in most instances, an irreversible lesion characterized by inability of the pancreas to produce sufficient quantities of insulin for his needs. The damage has already been accomplished. The factors responsible during the preceding months or years for inciting the vicious cycle which led to pancreatic damage may no longer be present. The time has passed when these factors might have been studied or when the possibility existed for breaking the cycle. The etiology of diabetes must be studied before the existence of the disease. Per se, this poses a problem of the first magnitude.

A possible alternate approach to the study of the etiology of diabetes mellitus in man lies in the recent demonstration that temporary diabetes mellitus can be produced experimentally in normal human beings.[7,8] The classical animal experiments of Houssay, Best, Young, Long, Lukens, the Coris, and others have indicated the importance to normal carbohydrate metabolism of a delicately integrated balance of functional activities of the pituitary gland, adrenals, thyroid, and pancreas. Permanent *pancreatic* diabetes has been produced in healthy animals by administration of suitable amounts of crude extracts of the anterior pituitary gland,[9] by prolonged and severe hyperglycemia induced by oft repeated intraperitoneal injections of glucose,[10] and by the necrotizing effect upon beta cells of injections of alloxan.[11]

Temporary diabetes in normal men and women has been attained by injections of purified preparations of pituitary adrenocorticotropic hormone. From the increasing severity of the diabetic state with increasing duration of such treatment, it seems clear that permanent pancreatic diabetes would eventuate were these injections continued beyond several weeks. In the early period of induction of diabetes in man one has an opportunity to study some of the metabolic de-

rangements which occur prior to the development of the state of permanent diabetes.

Studies in this period have disclosed several interesting facts. A disturbance of purine metabolism, the exact nature of which is still unknown, occurs during the induction of diabetes in man with adrenocorticotropic hormone. The level of circulating glutathione falls.[8] These two phenomena associated with the beginning of diabetes are reminiscent of changes known to occur when alloxan, conceivably a purine metabolite, is used to induce degeneration of beta cells in normal animals.[11] The suggestion has been made that excessive amounts of an alloxan-like substance may be produced during the induction of experimental diabetes in man.[8]

The decrease of blood glutathione affords interesting possibilities for future investigations. Reduced glutathione with its free sulfhydryl group, —SH, is ubiquitous in all cells of the body. Many enzyme systems are dependent upon —SH groups for their normal function. A decreased intracellular availability of —SH could affect metabolic processes profoundly by changing the normal activities of those enzyme systems dependent upon —SH. This may be of particular importance in beta cells, since their function is unique in that they produce a product, insulin, which is unusually high in the sulfur-containing amino acid, cystine. Cysteine is one of the three amino acids contained in glutathione.

It seems quite significant that when reduced glutathione is given intravenously during the induction of diabetes in man, glycosuria disappears abruptly and the blood sugar level falls.[8] These effects are transitory, however, lasting for but an hour or two following the administration of glutathione. The diabetogenic effect of the adrenocorticotropic hormone then becomes evident again, as indicated by a return of glycosuria and hyperglycemia.

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Diabetes in America: Current Problems and Needs

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Prepared for Modern Medicine

THE increasing importance of diabetes mellitus in the American population constitutes a challenge to American physicians. The scope of the problem has been demonstrated repeatedly, first by the remarkable rise in the mortality rates from diabetes, and second by a series of surveys which clearly indicate the large number of persons in the community who have diabetes without knowing it.

Although in 1929 a survey of chronic diseases by Bigelow and Lombard in Massachusetts had shown a surprising number of diabetic people in the population, the National Health Survey of 1935, conducted by the National Institutes of Health, indicated an incidence of diabetes of 3.67 for every 1,000 persons. Surveys during and especially since World War II have not only emphasized the large number of persons with diabetes, but have demonstrated that nearly half those with the disease are unaware of their condition. Through state and county medical societies, physicians must attack the problem more and more energetically.

SIZE OF THE PROBLEM

Diabetes is a major chronic disease. As a cause of death it ranks seventh among the diseases, if deaths from stillbirth and violence are omitted. Diabetes may occur at any age but is far more common after forty.

There are probably two million diabetics in the United States. Of these about one million have been diagnosed and most of these are under treatment. The number of potential diabetics is even greater. More than four million or about 3% of our population may eventually become diabetic. Spiegelman and Marks concluded that during the next few years:

> The number of diabetics in the United States will increase at a much greater rate than the total population. From 1940 to 1950 an increase of 18 per cent may be expected in the number of diabetics while the total population is expected to grow by only 9 per cent. At least 50,000 persons in the United States became diabetic during 1940, of whom two-fifths were male and three-fifths were female. Not quite half were under 55 years of age at the onset of the diabetes. Of our population under the age of 50, over 4 per cent of the females and more than 2 per cent of the males will eventually become diabetic (*Am. J. Pub. Health* 36:26, 1946).

Since the introduction of insulin, deaths from diabetes before the age of fifty have declined markedly. This decrease has been most prominent in the mortality of diabetic children. Diabetes is unique among the diseases common to middle and later

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life in that a specific means of control —insulin—is available.

MAJOR OBJECTIVES

To discover and treat the one million unknown diabetics is a prime task for American medicine. The use of laboratory methods makes diagnosis possible before characteristic symptoms have developed. Among the draftees studied at the Boston Induction Center during World War II diabetes could be demonstrated more than two and one-half times as frequently as expected.

It remained, however, for a survey by the U.S. Public Health Service to show that in a typical American community there are almost as many undiagnosed as diagnosed cases of diabetes. The survey was conducted in Oxford, Mass., a town whose population was a fair sample of the American population in age, sex, and nationality.

Among 3,500 persons whose blood sugar and urine were tested, 40 already were known to have diabetes. In addition, 30 other diabetics were discovered whose cases had been previously undiagnosed.

These studies confirm what physicians have long suspected: Earlier detection of the unrecognized diabetes patients in the community may reasonably be expected to prevent acute and dangerous complications, such as acidosis, coma, and severe infections, that so commonly develop in untreated cases. Moreover, the earlier use of insulin and diet may postpone the dreaded degenerative diseases which in the past have crippled so many patients who had had inadequately controlled diabetes for long periods.

Moved by these facts, the American Diabetes Association undertook to initiate a Diabetes Detection Drive in 1948, designating December 6 through 12 as Diabetes Week. In various cities and states of the country, committees were appointed by county medical societies and local diabetes associations who undertook to offer blood and urine tests in their communities in an attempt to find the undiscovered diabetics.

As Chairman of the National Committee on Diabetes Detection, I can make a partial statement of the results, which indicate that the American medical profession could readily succeed in an effort to find the million unknown diabetic patients and bring them under adequate treatment. During that Diabetes Week and the period immediately following, more than 130,000 persons were tested for diabetes.

The results varied from one community to another, but actually between 1 and 2% of all persons tested proved to be diabetics. In many communities patients who already knew they had diabetes came in for checkups. Excluding these known patients, the results leave no doubt that when urine tests are followed up by blood tests about 1% of the adults in any series will show previously unrecognized diabetes.

In 1949, Diabetes Week will be observed October 10 through 16.

DIAGNOSIS OF DIABETES

The typical symptoms of diabetes are latent, and diagnosis of the disease should be made long before the following symptoms have made their appearance:

Loss of weight and strength in spite of a good appetite
Increased thirst
Frequent urination
Itching
Aching pains in the extremities
Visual disturbances

Patients can be discovered early if all relatives of known diabetic patients are tested for sugar in the urine once every six months. The following laboratory findings indicate diabetes with a high degree of probability:

Any amount of glucose in the urine, accompanied by blood sugar values (mg. per 100 cc. blood) above the following levels:

1] Venous blood sugar
   a] By Folin-Wu method, over 130 fasting, over 200 after eating.
   b] By Somogyi method or other method indicating true glucose value, over 110 fasting, over 150 after eating.
2] Capillary blood sugar
   a] By Folin-Malmros method, over 140 fasting, over 240 after eating.
   b] By methods indicating true glucose value, over 120 fasting, over 200 after eating.

When the reports are close to these values or when there is a complication (for example, hyperthyroidism), repeated blood sugar tests should be secured or a glucose tolerance test performed for verification after the illness has subsided.

The sugar tolerance test should be done under normal conditions of diet; the diet for three days in advance of the test should include at least 250 gm. of carbohydrate per day.

Diabetes should not be diagnosed on the basis of one high blood sugar report alone. Diagnosis of diabetes may usually be ruled out if the blood sugar, in either capillary or venous blood, is less than 120 mg. per 100 cc. within thirty to ninety minutes after a meal which contains the normal amount of carbohydrate, providing the diet has not been restricted.

In diabetes detection efforts, large numbers of individuals can be tested by using either the Galatest or the Clinitest. Patients should be instructed to collect urine within ninety minutes after a meal containing a normal amount of carbohydrate, for example, an ordinary serving of potato, a slice of bread, a vegetable, and a simple dessert. Attempts should be made to test the urine of everyone in the community once a year and especially of relatives of known diabetics, obese individuals, persons over forty years of age, and any individual who feels below par.

EARLY, ADEQUATE THERAPY

Insulin and other measures in the management of diabetes when applied early and continuously make possible a normal level of health and activity. Nevertheless, many diabetic patients fail to get the best that science affords because they do not use insulin until years have passed and do not apply modern knowledge of diet. Repeated instructions are essential. Furthermore, continued attempts should be made to provide postgraduate courses and to arouse the interest of the general practitioner in diabetic treatment.

Insulin should be used as soon as the diagnosis is made in all young patients. There is no such thing as adequate treatment of a diabetic child or young diabetic adult without insulin.

IMPROVEMENTS IN THERAPY

Improvements in insulin have been sought in the direction of prolonging

its effects and of combining rapid and prolonged action in a single dose. Protamine zinc insulin acts for twenty-four to thirty hours after injection, depending somewhat upon the size of the dose. Thus, in a given individual, if a small dose of 20 units is employed, the blood sugar curve may indicate the action is twenty-four to twenty-six hours. With this same patient, a large dose of 40 to 50 units may be followed by a fall in blood sugar, indicating that the period of action is nearly thirty hours.

Crystalline insulin or regular insulin acts only six to eight hours. Other forms of insulin, notably globin, have a somewhat shorter period of action than protamine zinc insulin but a decidedly longer one than regular insulin.

In an effort to secure a form of insulin with both rapid and prolonged action, Dr. Hagedorn has described NPH-50. This type, prepared by crystallizing protamine insulin, has already been tried in a considerable number of patients but is not yet on the market.

The action of any insulin is improved if the patient's diet is suited to his needs. The diet should provide adequate minerals, sufficient vitamins, a proper caloric quantity, and balanced proportions of carbohydrate, protein, and fat. The more carefully and meticulously the diet is planned for a new patient and the more constantly it is followed, the better will be the results.

For patients whose diabetes begins in childhood, the onset of arteriosclerotic complications after fifteen years of the disease is common. Retinal hemorrhages, albuminuria, and hypertension frequently develop. However, in a series of such childhood diabetics followed over twenty years, the frequency of retinal hemorrhages and albuminuria was only one-fifth as great in those whose diabetes had been kept under good control as in those who, although still living, had not followed a diet, had not attempted to keep the urine sugar free, had not had frequent blood sugar tests, and had not escaped acidosis and continuous hyperglycemia.

Although it is not clear what factors underlie the premature aging and arteriosclerosis found so universally in diabetic patients, especially in youth after long duration of the disease, evidence at present indicates that earlier use of insulin, more faithful adherence to a proper diet, and frequent checkups by a physician prevent or postpone these complications.

ESTIMATION OF BASAL METABOLIC RATE when apparatus is not available is simplified by use of an index calculated by multiplying the respiration rate by the pulse rate. The basal metabolism rate computed by the Krogh method and the RP index corresponded within +15% to −10% in each of 100 patients investigated by Eugene Bene, M.D., Prague. The patient should lie quietly for five minutes before counts are done and should be unaware that his respiration rate is being taken.

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Diet in Diabetes

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Prepared for Modern Medicine

THE treatment of diabetes mellitus is based upon the adjustment of diet, insulin, and exercise to the individual patient. Diet is the foundation of treatment. Insulin from a source outside the body is employed when necessary. Exercise is a factor which in optimal amount enhances the use of food by the body and increases the efficiency of the influence of insulin.

The discovery of insulin 28 years ago made it possible for patients with diabetes to have diets more nearly approaching those of healthy persons. In general, any agents which enable the physician to restore the patient's body to a status approaching normal will insure the most satisfactory results in the treatment of diabetes.

DIET COMPONENTS

A few years after the discovery of insulin, the *carbohydrate* fraction of the diet for patients with diabetes was gradually increased until normal or above normal quantities were employed by some. At present, however, the pendulum has swung backward to a middle ground where the average diet calls for approximately two-thirds of the carbohydrate commonly employed in a normal diet. In special instances, particularly when heavy physical activity is required, this proportion may be exceeded.

Likewise, in recent years, with advances in knowledge of protein metabolism, liberalization of the *protein* component of the diet has been rather generally accepted. Present-day diabetic diets call for more protein than was commonly employed in the past. When carbohydrate and protein have been allocated, *fat* is employed to complete the desired total calorie value.

BASAL REQUIREMENT

The total calorie value of the diet may be established by the following computation: From available tables, the number of calories for the basal requirement, the calories required at rest, may be determined. This computation is based upon the height and age of the patient and the average normal weight for height and age, or whatever may be considered the optimal weight for the patient. Then, by adding one-quarter to one-half, depending upon the activity of the patient, the approximate number of calories for maintenance are determined. Special allowance must be made for growth in children, varying with age.

Maintenance of optimal weight, plus a sense of well-being and near

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TABLE 1. FIXED WEIGHED DIET

Diet order: C 165 P 80 F 90 *Carbohydrate distribution:* B 50 L 50 D 50 BT 15

		Grams	*Carbo-hydrate*	*Protein*	*Fat*
Breakfast					
one only	5% fruit or	200			
	10% fruit or	***100***	10	1	
	15% fruit	65			
one only	Cooked cereal or	***135***			
	Dry cereal	27	20	2	
one only	Egg or	***one***		6	6
	Bacon, crisp (plus 1 egg)	10			
	Ham, lean	25			
	Bread	30	15	3	
	Butter	10			9
	Milk	60	3	2	2
	Cream, 18%	30	1	1	6
	Coffee or Tea as desired				
	Total		49	15	23
Luncheon					
	Soup (clear broth occasionally)				
one only	American cheese or	***45***	Deduct 7 gm. butter		
	Cottage cheese or	70			
	Meat, medium fat	50		14	9
one only	3% vegetable or	***200***			
	6% vegetable	100	6	1	
	*Bread	40	20	4	
	Mayonnaise or Butter	15			13
	Milk	200	10	6	8
one only	10% fruit or	150			
	15% fruit or	***100***	15	1	
	20% fruit	75			
	Total		51	26	30
Dinner					
one only	Shrimp or	105	Add 15 gm. butter		
	Crab or Lobster or	125	Add 15 gm. butter		
	Fresh white fish or	110	Add 10 gm. butter		
	Salmon or	95			
	Tuna or	90	Deduct 5 gm. buttter		
	Meat, medium fat	***75***		21	14
one only	3% vegetable or	200			
	6% vegetable	***100***	6	1	
one only	15% vegetable or fruit or	135			
	20% vegetable or fruit	***100***	20	3	
	Bread	30	15	3	
	Mayonnaise or Butter	10			9
	Cream 18%	30	1	1	6
one only	5% fruit or	200			
	10% fruit or	***100***	10	1	
	15% fruit	65			
	Total		52	30	29

TABLE 1. FIXED WEIGHED DIET *(Cont.)*

	Grams	Carbohydrate	Protein	Fat
Bedtime (10 p.m.)				
Milk	200	10	6	8
and				
Crackers or	7	5	1	
Ovaltine or	1 diabetic recipe**			
Chocolate	1 diabetic recipe***			
and				
Crackers	7			
Total		15	7	8
Breakfast		49	15	23
Luncheon		51	26	30
Dinner		52	30	29
Bedtime		15	7	8
Total Diet		167	78	90

*In place of 40 gm. of bread, use 100 gm. of 20% vegetable or fruit.

**Hot Ovaltine (1 diabetic recipe)

		C	P	F
Milk	180 gm.	9	6	7
Ovaltine	7 gm.	6	1	
Saccharin	1 quarter-grain tablet			
Vanilla	2 drops			
		15	7	7

Scald milk. Mix a small amount with the Ovaltine to make a paste. Add to the scalded milk. Remove from fire and add saccharin and vanilla.

***Hot Chocolate (1 diabetic recipe)

		C	P	F
Milk	180 gm.	9	6	7
Unsweetened cocoa	3 gm.	1	1	1
Saccharin	2 quarter-grain tablets			
Vanilla	1 to 2 drops			
		10	7	8

Diets prepared by Miss June Pettibone, Dietitian, Mason Clinic, Seattle.

TABLE 2. SAMPLE MENU

Breakfast	Grams
Orange juice	100
Oatmeal	135
Egg, soft cooked, one	
Bread, toasted	30
Butter	10
Milk	60
Cream, 18%	30
Coffee or Tea as desired	
Luncheon	
Sandwich:	
American cheese	45
Bread	40
Lettuce leaf	
Butter	4
Mayonnaise	4
String beans	
3% vegetable salad with vinegar dressing:	
Lettuce and Celery	
Sliced tomato	100
Milk	200
Fresh apple	100
Dinner	
Roast beef	75
Diced carrots	100
Baked potato	100
Bread	30
Butter	10
Cream, 18%	30
W. P. Pears	100
Tea or Coffee as desired	
Bedtime (10 p.m.)	
Milk	200
Crackers, soda	7

normal efficiency and endurance, constitutes the yardstick for determining the adequacy of the diet from a calorie and nutritional standpoint. Only the amount of food necessary to accomplish the foregoing should be employed, and insulin from an outside source, if required, must be adjusted to this necessary diet, so that the food is almost totally used by the body and not lost as urinary sugar.

Control of the diabetes while the patient consumes the necessary diet, as indicated by sugar-free urine and blood sugar levels approximating normal, gives the best assurance of maintaining current health and protecting future welfare.

DIET ARITHMETIC

Since all phases of adequate nutrition must be given consideration, variety of food is important. The basic necessary foods must be incorporated in the menu. Fruits, vegetables, cereals and milk, and different sources of protein—meat, fish, fowl, cheese, and eggs—must be emphasized. Thus, requirements concerning vitamins, minerals, bulk, and so on, will be met.

To know and follow the diet, the patient must understand diet arithmetic and must develop ability to estimate food quantities. The arithmetic is simple and does not demand higher education, but it does require a little study and application by the patient. The quickest and most accurate method of acquiring the ability to estimate food quantities is by food scales.

In diabetes clinics, diet arithmetic and quantity estimation may be taught in different ways, but whatever method used, total calorie value and sugar value of the diet may not vary 10% above or below the optimal from day to day if satisfactory control of the diabetes and balance between diet and insulin are to be maintained. Therefore, daily constancy of these two phases of the diet is important.

It is not within the scope of this article to present details of diet arithmetic. Many excellent books for the patient and physician are available as well as class and individual instruction in any teaching diabetes clinic. The American Diabetes Association, 1 Nevins Street, Brooklyn 17, N.Y., will soon have a diet manual giving the current composite opinion of authorities regarding the optimal diet of the diabetic.

SAMPLE DIET PLAN

The dietitian plans a fixed diet by weight (Table 1) for each patient. The prescription ordered by the physician, less the value of interval or bedtime feedings, if prescribed, is divided between three meals.

A bedtime feeding of milk nourishment is included for the patient who is taking protamine zinc insulin. The carbohydrate for this feeding is subtracted from the total carbohydrate. The remainder is then divided equally between the three meals. Some protein is included with each meal, and the fat is divided as needed.

Since it is difficult to figure the carbohydrate for each meal to the gram, a variation of 2 gm. in either direction is allowed. It is also impossible to figure the total diet exactly as prescribed; therefore a variation of 5 gm. above or below the prescribed

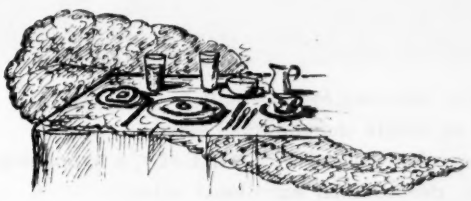
amount of carbohydrate, protein, and fat is allowed.

In using the fixed weighed diet plan, the patient decides what foods he wants to eat, determines from the table the percentage group for the fruits and vegetables, and then weighs the amount indicated in the grams column. Though he is taught the food value of plain foods, he need not figure the carbohydrate, protein, and fat as a daily routine.

The sample menu in Table 2 shows how the diet plan is used in meal planning. The groups from which this sample is chosen are indicated in italics in the gram weight column.

Special foods are not necessary. The food items incorporated in the menu should be all the useful foods employed for sustenance by human beings, except sugar and foods prepared by the addition of sugar. Sugar is such a simple carbohydrate that it presents a rapid demand upon the disabled sugar mechanism of the body in contrast to the gradual demand made during digestion when certain fractions of the total diet are slowly converted to glucose.

A normal variety of foods is not only possible but desirable for the diabetic. The total necessary calories must be employed and not exceeded. What a contrast this is to the situation twenty-five or thirty years ago!



SUBCUTANEOUS IMPLANTATION OF INSULIN has been used with good results in treatment of 7 cases of diabetes, reports Luis Vargas, M.D., of the Catholic University of Chile, Santiago. Pellets of protamine zinc insulin cholesterol were implanted subcutaneously. The dosage ranged from 20 to 92.7 units of insulin per kilogram of body weight, except in 1 case in which 450 units were given. The pellets are absorbed as slowly as fat-soluble hormones and are active up to one hundred days. Although initial results are promising, evidence is lacking that the rate of insulin release is influenced by the level of the blood sugar and the secretions of the pituitary or thyroid. Another difficulty is the impossibility of standardizing treatment, since the fluctuating diabetic state often demands that the dosage of insulin be changed at short intervals.

Lancet 256:598-601, 614-615, 1949.

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*Levine, S. Z.: J.A.M.A. 128:283, May 26, 1945.

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The Insulins in Diabetes

FRANKLIN B. PECK, M.D.*
Indianapolis General Hospital, Indianapolis

Prepared for Modern Medicine

THE action of insulin is still best described in terms of what it does for the diabetic patient. When given in appropriate doses he is enabled to utilize foodstuffs in a comparatively normal manner, the blood sugar concentration is confined within normal limits, the urine remains free of sugar and ketone bodies, and acidosis and coma are prevented.

These actions of insulin are inherent in all active preparations of the hormone, including the numerous modifications that have been described, but the rates of insulin effect differ in the various preparations. Regular insulin and that made from zinc insulin crystals are rapid in action and effective over a period of five or six hours or more. Globin insulin with zinc has an intermediate duration of action considered by most observers to reach its maximum in about eight hours and extending to sixteen or eighteen hours. Protamine zinc insulin acts well beyond a twenty-four-hour period and, if given in large doses, may extend its effect into the third day. The rate of onset and duration of effect of any insulin preparation vary greatly in different individuals and in relation to the size of the dose.

For many years, and especially since the work of Allen, Stillman, and Fitz[1] on the total regulation of the diet, the intake of food has come to be regarded as the metabolic load which the diabetic economy is incapable of properly metabolizing without the injection of exogenous insulin. It was natural to adapt the use of the original short-acting insulin preparations to conform with the intervals shown by high blood sugar levels and sugar output to be efficacious in preventing wastage of energy-producing substances in the urine. Clinical experience dictated the spacing of insulin injections in relation to the main food loads of the day, and in general the daytime or feeding hours were well controlled although several injections per day were necessary in severe cases.

A major difficulty arose during the fasting hours at night and in the early morning when the severe case, as exemplified by the juvenile diabetic, commonly was acidotic before breakfast and for an hour or two thereafter. Hepatomegalia was almost the rule and the pot-bellied diabetic dwarf was a common clinical sight. These difficulties melted away dramatically the first year after protamine zinc insulin came into general use, even in children who were not continuously sugar free.

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Thus the protection of the patient from the ravages of uncontrolled active diabetes during the night has been the great contribution of protamine zinc insulin, and this is only accomplished by a preparation which acts beyond twenty-four hours. It is an effect that must not be sacrificed even at the expense of some daytime glycosuria or multiple injections.

There is something specifically beneficial to patients in the maintenance of normal metabolic processes over the nocturnal hours of starvation. Is the glycosuria that occurs over this period more hazardous to the patient's tissues and vascular system than the glycosuria following meals? Nocturnal hyperglycemia and glycosuria usually result when there is an inadequate insulin effect over the night and the origin may well signify a basic failure in body synthesis[2] and an onset of destructive processes, whereas daytime glycosuria may represent merely an overflow mechanism from entirely exogenous sources. Surprisingly little attention has been given this possibility.

Wilder[3] in 1937 demonstrated the protective effect of protamine zinc insulin against nitrogen wastage even though a persistently high blood sugar level and glycosuria were intentionally maintained. More work should be done on this point. It is stressed here in order to emphasize an apparently crucial factor in choice of an insulin effect obtained by a single daily injection; that effect should be long enough to insure the patient against hyperglycemia and glycosuria during fasting. This can be determined by the height of the blood sugar and the urine sugar tests before breakfast. If these are not within reasonably normal limits, the effect of the insulin preparation is too short and another combination capable of longer action should be substituted.

The daytime levels obviously serve as criteria for prescribing larger or smaller amounts of rapidly acting insulin. Mild cases and some moderately severe ones respond well to a single injection of protamine zinc insulin in doses up to 20 or at most 30 units per day. Beyond this range of dosage the daytime food load usually necessitates additional insulin, either as a supplemental dose or as an addition to a mixture, thereby "solubilizing" protamine zinc insulin.

In this discussion the term "insulin mixtures" refers either to the actual combination in the syringe of desired quantities of standard preparations of insulin and protamine zinc insulin, in which case the combination is called *extemporaneous,* or to a combination made in bulk in the laboratory or in sterilized vials, in which event it is usually referred to as *premixed.* Extemporaneous mixtures are ordinarily made just prior to injection and the proportions are therefore flexible and subject to alteration to fit anticipated conditions, whereas bulk premixtures are necessarily fixed in their proportions and in time activity. Premixtures can be altered only by further additions of insulin or protamine zinc insulin to the vial.

There has been considerable controversy over the respective merits of these two procedures, although their fundamental difference—adjustability as opposed to fixed time activity—would seem sufficiently to delineate their uses and indications for the in-

dividual case. If a fixed premixed combination is chosen, the clinician or a trained technician must make the mixture under proper aseptic conditions, usually by mixing the contents of standard vials obtained on the market in a large (50-cc.) sterile syringe and reinjecting the mixture into the original vials. Premixtures will keep for a few months in a cold place.

Mixtures provide effects that represent a transition or blending of the action of short- and long-acting insulin preparations. To describe their effects as intermediate is not enough, for they may act within almost any range between the standard actions of insulin and protamine zinc insulin. The early concept that mixtures possessed both the characteristic abrupt but short effect of insulin and the slow but prolonged action of protamine zinc insulin has been abandoned in favor of the view that they provide an insulin effect of almost any degree of duration depending upon their overall rates of solubility and release of active principle from the depot in the tissues. Duration is achieved at the expense of rapidity of action, and intensity is sacrificed for length of effect.

It has been amply demonstrated[4] that the slow but prolonged action of protamine zinc insulin can be progressively shortened and correspondingly intensified by mixing the preparation with proportionately greater quantities of unmodified insulin. The more insulin added, the more intense and less prolonged is the effect. There is not much to be gained therapeutically unless at least an equal amount of insulin in units is added to the protamine zinc insulin.

The most generally useful combinations have ranged from 3:2 to 3:1 parts of insulin to protamine zinc insulin respectively, with the 2:1 mixture serving optimum needs in about 80% of cases.[5] In my experience the combination of 3 parts of insulin to 1 part of protamine zinc insulin acts rather similarly to globin insulin with zinc and in the average severe case does not quite span the early morning hours unless large doses are given. A 3 P.M. feeding is necessitated to prevent hypoglycemia in late afternoon.

The 2:1 mixture usually lasts long enough to maintain satisfactory fasting levels and, on account of its moderate overlap from one day to the next, the daily morning doses reinforce one another sufficiently to carry the postprandial breakfast period adequately. This period, however, usually has the highest level for the day, and occasionally there may be need for further dietary redistribution by lowering the food value for breakfast and adding it to other meals. Only in exceptional circumstances has it been necessary to use the 3:2 combination in order to obtain sufficient nocturnal effect, but one should not hesitate to do so, or even to mix equal parts of insulin and protamine zinc insulin, if necessary.

Insulin mixtures should not be expected to solve all the problems of insulin therapy. They provide only a means of applying an almost unlimited range of insulin time activities, which may be varied at will from an effect as long as that of protamine zinc insulin to one as short as that of unmodified insulin. Obviously no mixture, whether made in syringe or vial, can automatically meet the require-

ments of the rapidly shifting complicated case, the "brittle" insulin-sensitive patient, or the individual whose dietary intake, emotional state, and exercise fluctuate unpredictably from day to day or during the day.

The single-dose procedure using mixtures or other modifications is most ideally suited to the patient with relatively stable insulin requirements. Nevertheless, even in unstable cases, the results obtained are as good or better than those obtained by other methods, and allow some freedom from multiple injections.

It is usually advisable to begin treatment of the complicated case or the more severe new case with protamine zinc insulin and unmodified insulin in separate supplementary doses. The following method is suggested for recent uncomplicated cases and for those not previously treated:

1] The first day give 10 units of protamine zinc insulin.

2] Increase daily by 5 to 10 units until urine is nearly sugar free on arising or until the fasting blood sugar approaches normal levels.

3] If glycosuria and hyperglycemia persist after meals, add supplementary insulin (varying with Benedict test), e.g., *yellow*—5 units; *orange*—10 units; *red*—15 units.

4] Replace multiple injections with single-dose admixture of 2 parts insulin to 1 part protamine zinc insulin. The criteria for adjustment of admixture are identical with those governing separate doses. Mixtures are always made from preparations of the same concentration.

a] If daytime glycosuria and hyperglycemia persist, increase insulin content.

b] If postabsorptive (fasting) glycosuria and hyperglycemia persist, increase amount of protamine zinc insulin.

The dose of protamine zinc insulin is gradually increased until satisfactory levels are maintained before breakfast. Supplementary doses of regular insulin are prescribed according to the findings in the daytime. When conditions have become reasonably constant from day to day the total dosage is combined into a 2:1 mixture in the syringe and given as a single dose before breakfast.

Further readjustments may then be made depending upon the levels and glycosuria in the fasting and postprandial periods. More protamine zinc insulin is indicated for greater effect on the fasting levels, and more insulin is needed if the daytime sugar wastage is excessive.

The necessity of teaching the patient to mix insulin preparations has been regarded by some as a disadvantage of this technic. Certain patients are incapable of learning to give even a single dose of unmixed insulin dependably and accurately. In those with failing vision or other physical disabilities the same problem arises. In such instances the premixed mixtures in a vial are very helpful.

Much study has been given to the preparation of ready-made mixtures and modifications having similar attributes. A few have shown considerable promise, although none are as yet available. Even should such a modification be marketed, instances will undoubtedly remain in which a more adjustable effect is of advantage and where supplements of rapidly acting insulin will be occasionally necessary. Nevertheless, a modification having the time activity of a 2:1 mixture should be adequate in a single daily dose for about 80% of the patients about 80% of the time.

(References on page 110)

Diabetes in Children

HENRY J. JOHN, M.D.*
Cleveland

Prepared for Modern Medicine

TODAY, diabetes in children is a relatively common medical problem. This was not the case as recently as thirty years ago, for then the child with diabetes did not survive long—a few months, occasionally a year or two. Because all children who contracted diabetes were doomed to an early death, there was no accumulation of cases into a sizable group until the discovery of insulin by Banting and Best in 1921 made it possible to treat diabetic children and prolong their lives.

Even though we now have a considerable number of these children to deal with, the incidence of diabetes in childhood is still relatively low compared to that of diabetes as a whole. In my series of 6,000 diabetics, only 4.5% were in the first, and 4.16% in the second decade of life when first examined. The majority of patients are past the fourth decade.

EARLY INSULIN PERIOD

After the disheartening experiences with diabetic children in the preinsulin period, the early results of insulin treatment were most encouraging. We saw them regain their best weight and their lost energy and, for all practical purposes, lead the lives of normal children.

It seemed for awhile as though the problems presented by juvenile diabetes had been almost completely solved, because these children, with but few exceptions, grew and developed normally, did well in school, went to college, graduated with honors, and began to fill important positions in their communities. It was a common observation that diabetic children as a group were above average mentally, a compensating factor for their handicap.

LATER COMPLICATIONS

This optimistic attitude continued for nearly two decades, then less encouraging signs began to appear. After ten or more years of diabetes many young persons began to develop arteriosclerosis. Many had diabetic retinitis with or without hemorrhage into the retina, some with complete blindness owing to detachment of the retina. Serious kidney involvement such as intercapillary glomerulosclerosis occurred in others and led to death.

These complications have altered considerably our ideas regarding the future of the diabetic child. And as yet we do not know what to do about preventing or controlling these conditions, which appear not only in children and young adults who have had inadequate medical care, but also in those who have had good care.

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Dolger has reported that in a group of 55 patients who had had diabetes for twenty-five years, retinitis with hemorrhages occurred in all. Of these, 16 had diabetes before the age of ten and 39 first had the disease between the ages of ten and twenty. Others have reported similar findings. Joslin and White state that arteriosclerosis was revealed by roentgen examinations in 92.3% of patients whose diabetes began in childhood and persisted for twenty years or more.

The incidence of intercapillary glomerulosclerosis also is growing. A high-protein diet has been advocated for these conditions and seems to be of value, but more time must elapse before we shall be able to judge the results fully. The diet should be tried in all cases, since we have nothing else to offer. The use of rutin, which gave some promise as a treatment for diabetic retinitis, is now, after more than a decade of experience, considered of little value by many ophthalmologists.

Hence it is now obvious that, although insulin gave life and comparative health to diabetic children and allows them to grow and mature normally, the problems of juvenile diabetes are still far from solved.

What can reduce the number and severity of cases of juvenile diabetes? Why do children develop diabetes?

A look at the facts shows that sex is not a factor affecting the appearance of diabetes in children. In adults the incidence in females predominates, but in children the incidence is practically equal for the two sexes. In my own group of 500 children, 51% were boys and 49% girls. In 6,999 cases gathered from the literature, 49.5% were boys and 50.5% were girls.

ETIOLOGY OF DIABETES IN CHILDHOOD

Two etiologic factors do stand out prominently: heredity and infection.

Heredity—Evidence of a hereditary factor is found in almost one-third of diabetic children. Heredity is more important in juvenile than in adult diabetes because in children we deal with pure diabetes—the degenerative processes such as hypertension, arteriosclerosis, hyperthyroidism, hyperpituitarism, hepatitis, and so forth, that influence the incidence of diabetes in adults, are absent.

In my series of 500 children with diabetes, I found evidence of a hereditary factor in 31.5%. In other reports in the literature, figures range from 11.6 to 54%. However, some of these may not be completely accurate or tell the whole story, for it has been my experience that the longer one studies children with diabetes, the more the disease shows up in their families as the years go by. Repeatedly in my own cases there have been instances with no trace of diabetic history in the family at the time of the patient's first examination, and yet a few months or a few years later I will be treating the child's father or uncle for diabetes.

If the records are to be kept accurate regarding heredity, it is necessary to ask periodically, "Are there any new cases of diabetes in your family since I last saw you?" With additional information gained in this way, I would venture an estimate of hereditary tendency to diabetes in 40% of these children. The incidence of hereditary factor is slightly higher in

Jewish than in gentile children. In my series of 500, it was 31.3% for the gentile and 35.5% for the Jewish children.

Infection—The other prominent factor affecting the development of diabetes in children is infection. In a certain proportion of cases, the childhood diseases, such as measles, mumps, scarlet fever, tonsillitis, influenza, and pneumonia, are followed by diabetes, often in a few days or within a month or two. I have reported 122 patients of my own who developed diabetes within sixty days after an infection and 61 cases of the same type described by others, a total of 183 cases. I have observed other cases in which diabetes developed within ninety days after an infection.

We do not yet know why infection precipitates diabetes in 1 child and does not do so in 99 others. One can merely speculate as to the reason. Is it that the child was born with but a small insulinogenic reserve which is easily exhausted by the additional strain of an infection? Or is it perhaps that in the individual case a particular infection causes more virulent toxemia?

There is no doubt that infection affects the function of the pancreas adversely. In a diabetic child under good control on, for instance, 20 units of insulin a day, a febrile infection will upset the condition quickly. It may require 80 units to control the increased hyperglycemia and prevent severe acidosis and probable coma.

We can well picture a similar situation in a nondiabetic child during an infection. Even a normal child often has some rise of blood sugar during infection. There are several possible explanations. Toxemia is produced, which tends to block the action of insulin. Thus, though the same amount of insulin enters the blood stream from the pancreas, part of it is blocked and the blood sugar is increased, just as if the pancreas had not produced enough insulin.

Another possibility is that there may be an increased output of some internal secretion from the pituitary, adrenal, or thyroid gland. The effect of all these secretions is similar in interfering with the action of insulin, though in each instance the mechanism is different. Any interference with the action of insulin increases the blood sugar.

Still another possibility is that the toxemia may exert a direct effect on the islands of Langerhans and decrease secretion of insulin, thus bringing about hyperglycemia. Just which of these possible mechanisms, alone or in combination, is at work cannot be determined at present for we have no laboratory methods to measure the functional activity of these glands.

In view of the many cases of juvenile diabetes which appear following the acute exanthems, it should be the rule to examine the urine for sugar once a week for four to six weeks after such an infection. In this way, diabetes would be discovered early, with great advantage to the child. The process affecting the islands of Langerhans may still be reversible, whereas in later stages the damage becomes permanent. This does not mean that a cure can be accomplished by early diagnosis. The insulinogenic functional weakness is there and will remain, but the earlier its recognition, the better the chance to control the condition and prevent further damage.

MORTALITY OF DIABETIC CHILDREN

How long do children survive after diabetes develops? In my own group of 500 children, 136 have been lost to follow-up. Of the remaining 364 patients, 303 are living and 61 (16.7%) are dead. Of those living, 41.25% have had diabetes for ten years or less, 43.23% have had diabetes for ten to twenty years, and 15.31% have had the disease for over twenty years. These percentages no doubt will change with time, and those who have had the disease for twenty years or more will probably survive for a much longer time.

Diabetic coma was the most frequent cause of death in this series. It accounted for 31, or 50.8%, of the deaths. This is understandable, for the group was observed over a period of twenty-seven years, thus including a few cases seen before insulin was available and numerous others seen during the first years of insulin. We must bear in mind that in the first decade following the discovery of insulin, its use was largely experimental and not fully understood. In many instances, there was fear of using large enough doses. This fear played havoc in emergencies and accounted for the high mortality from coma in that period.

However, since the insulin routine has become well established and younger men have received practical training in it and are no longer afraid of it, gradual improvement has been evident from year to year. In time we shall see even better results. The best treatment for coma is prevention or, at least, early recognition and vigorous treatment by administration of large doses of insulin and of saline and glucose solution. Satisfactory response should appear within six to eight hours.

Treatment has improved tremendously in the last decade, but still is not what it should be. Death from diabetic coma was excusable in the early days of the insulin era, but it is somewhat disheartening that 2 recent deaths occurred in one of our best hospitals, where all facilities for adequate treatment were available.

The second most frequent cause of death in the diabetic children studied was *intercapillary glomerulosclerosis.* Of the 61 deaths, 11, or 18% of the total mortality, were the result of capillary glomerulosclerosis. The remaining deaths were from varied causes, some, such as accident, unrelated to the diabetes.

DIAGNOSIS IN CHILDHOOD

The diagnosis of diabetes in children rarely presents the difficulties it sometimes does in adults. In a child, onset usually is rapid and most of the classical symptoms are present from the start. Conversely, the onset of diabetes in adults is frequently insidious.

One pitfall to guard against is that of basing a diagnosis of diabetes in children on glycosuria alone. If heavy glycosuria appears together with most of the cardinal symptoms of diabetes, the diagnosis is practically a certainty, although there are exceptions. On the other hand, if only glycosuria is present, even though heavy, and the child does not have excessive thirst or urinary frequency, the assumption of the presence of diabetes is unwarranted and may prove to be a serious diagnostic error.

TREATMENT

For thirty years, I have followed the maxim: The sun should not set before treatment of a diabetic child is started. As soon as the diagnosis is established, which takes only an hour or two, treatment should start. My own practice is to give the child a dose of insulin in the office, before he goes to the hospital, so as not to lose any time.

Treatment consists of diet and insulin. It is always wise to start with insulin. If the case is mild, insulin can be discontinued later. Much is to be gained by immediate use of insulin and much to be lost by withholding it at the start. From the psychologic standpoint it is better to administer insulin at first and discontinue it later, if so indicated, than to lose time experimenting with low diets and then have to institute insulin treatment. Such a procedure undermines the confidence of the family and loses precious hours or days in the hospital, thus increasing the expense.

To start with a low diet, a starvation diet, in order to avoid insulin, is not rational therapy. The child is sick and has already lost much weight. To place him on a low diet to control glycosuria means a further loss of weight. This is undesirable and discouraging to the child as well as to his parents. It is bad psychology as well as bad medicine.

The diet should be liberal and calculated according to age. A child requires more calories per kilogram of body weight than does an adult, for he must compensate for extra activity and growth and usually for the weight loss incurred before the diagnosis was made. Wetzel's grid furnishes a reliable and scientific guide to the caloric needs of children of all ages.

In planning the diet, it must be remembered that if a child does not get enough food at home, he will secure it elsewhere. No child will go hungry for any great length of time. This fact we must anticipate and thus prevent the necessity of his stealing food at home or outside. Once this habit starts, it is hard to break.

Hunger in a diabetic child is not necessarily the result of inadequate food intake. It can also result from overdosage of insulin or improper distribution of the dosage. If insulin is not properly adjusted to the child's needs, hypoglycemia may result, which in itself creates extreme hunger. In such an instance, food is an urgent physiologic necessity and if it is not taken in time, the resulting insulin reaction may be quite severe and may require many more calories to counteract than would have been the case if food had been taken earlier.

Thus the stealing of food is not always the child's fault. Frequently, in fact a large part of the time, it is the fault of the physician. And when such a child is scolded and humiliated because of it, resentment is naturally created in him, for he knows intuitively that he is right and that the physician is wrong.

The handling of insulin reactions should be a problem for the family, not for the doctor. Instead of getting unduly excited and calling for medical help, which is rarely needed, the mother should be taught to act promptly. In this way she will lose the fear of reactions and will save both time and expense.

A mild insulin reaction can be counteracted by food in any form, a glass of milk with 2 or 3 tsp. of sugar, orange juice with sugar, hot water with sugar, ginger ale, candy, and the like. Karo syrup is better than sugar because it is unpurified glucose and does not need to be digested before it can be absorbed and enter the circulation. I always advise parents to keep Karo syrup on hand for such emergencies.

If the reaction has become severe because nothing was done earlier and the child is unconscious, the mother should give 0.5 to 1 cc. of adrenalin hypodermically, just as she gives insulin. This may counteract the insulin reaction by releasing glycogen from the liver, thus controlling the hypoglycemia. She should try this before calling a physician.

If adrenalin is not effective, the only thing that can be done is to administer 5 to 10 cc. of sterile 50% glucose solution intravenously, which of course must be done by a physician. This usually restores consciousness within a few minutes. It is wise for the family to keep 2 or 3 sterile ampules of 50% glucose at home, because the physician may not have it in his bag and much time may be lost.

Frequently, much too frequently, an insulin reaction is referred to as insulin shock. This is mistaken psychology and frightens the patient and the family before a reaction ever happens. Moreover, if proper emphasis is placed on precautions, the reaction may never happen. It is poor medical practice to speak of insulin shock and the other disagreeable possibilities of diabetes to the parents when first interviewed. They will not understand everything or remember it.

The parents are emotionally upset when they first learn that their child has diabetes. Hence the way should be made as easy as possible. They should be given encouragement and told only a few essential facts at first. At each visit, more detail should be added. If the instruction is presented in careful stages, they will eventually comprehend and understand the whole problem. Such management achieves much better results than an avalanche of instructions and admonitions at the outset.

DIMINISHED ANTIBODY PRODUCTION associated with diabetic complications such as gangrene and osteomyelitis appears to be related to serum protein values rather than to the fasting blood sugar levels. At Philadelphia General Hospital, 64 patients with diabetes were studied immunologically and chemically. Nineteen of the patients who had hypoproteinemia received lactalbumin or casein concentrate. Michael G. Wohl, M.D., and associates found that diabetic patients with hypoproteinemia have lower average agglutination titers than those with normal blood protein values, whereas capacity to produce antibody does not appear to be decreased in patients with hyperglycemia. Oral nitrogen supplementation enhances antibody formation.

Proc. Soc. Exper. Biol. & Med. 70:305-307, 1949.

Diabetes Complicating Pregnancy

PRISCILLA WHITE, M.D.*
Tufts College, Boston

Prepared for Modern Medicine

MANAGEMENT of obstetric diabetic patients begins in the prepregnancy period when these patients consult us about the following questions: first, the chances of a diabetic woman reproducing a diabetic child; second, the chances of her reproducing a living healthy child; and, third, the risk of pregnancy to the diabetic woman.

CHANCES OF DIABETIC CHILD

Based upon actual experience, the chances for a diabetic woman reproducing a diabetic are slight indeed. In our entire series of obstetric patients, only 2 children are classified as diabetics. One of these was so classified on the basis of a glucose tolerance test which deviated but a slight degree from normal. The other is the child of 2 diabetics.

The probability for the reproduction of a diabetic is explained upon the basis of the transmission of diabetes through mendelian recessive genes and the classification of the marital partner as a diabetic, a potential diabetic, or hereditary carrier of the disease. The potential diabetic (genetically so classified) is that rarely seen individual who is the offspring of 2 diabetics or the similar twin of a diabetic.

The chances of any individual being a carrier of diabetes are predicted as follows: 1 in 1 (100%) if the child of a diabetic; 1 in 2 if a grandchild; 1 in 4 if a great-grandchild; 1 in 8 if a great-great-grandchild. Our advice is carried out to the F_1 generation only.

FETAL DEATHS

The chances for fetal fatalities, in our experience, have been influenced by the following four factors: [1] poor control of maternal diabetes, [2] the degree of maternal vascular disease, [3] the occurrence of congenital defects, and [4] the imbalance of the sex hormones of pregnancy.

The maximum degree of poor control of diabetes—diabetic coma—coincided with 10% of our fetal fatalities. The most pernicious of the harmful effects was the degree of maternal vascular disease. In our experience it occurred in 40% of the fetal deaths. When arteriosclerosis progressed so far that the pelvic blood vessels were calcified, the fetal survival was only 10%. In a group of cases given all treatment which we believe is right and proper in the management of the obstetric diabetic patient, the fetal survival in patients with pelvic vascular disease rose to 40%.

The most tragic of the harmful effects, because it is unpredictable, is

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the occurrence of a congenital defect. Of the fetal fatalities, 10% occurred in relation to fetal defects. The total incidence of congenital defects in our experience is 80%. Among all the infants, however, only 10% had defects classified as major, that is, grossly disfiguring, incompatible with life or health, or requiring major surgery for correction.

These anomalies occurred most often in tissue which is bone or cartilaginous in origin. Included were syndactylia, clawhand, clubfoot, congenital dislocation of the hip, hydrocephalus, anencephalia, meningocele, defect of the skull, lesions of the eye, strabismus, retrolental fibroplasia, deaf-mutism, pyloric stenosis, hemangioma, and defect of liver, kidney, and heart. The most frequent of the harmful factors was the imbalance of the sex hormones of pregnancy, coinciding with 98% of the fetal fatalities and occurring in 80% of the patients observed.

MATERNAL DEATHS

The mortality and morbidity risk of the diabetic pregnancy to the diabetic woman parallels degree of vascular disease and consequent duration of diabetes. Among 500 cases there were 2 maternal deaths, both unrelated to the pregnancy. Twenty-five years' duration of diabetes, retinitis proliferans, and diabetic nephritis should be contraindications for pregnancy from a maternal point of view.

With pregnancy, so far as the diabetes is concerned, there is no evidence of increase in severity, but possibly a decrease in insulin requirement.

MANAGEMENT OF PREGNANCY

The dietary management of diabetes during pregnancy is planned to meet the increasing metabolic needs of the mother and the child. The insulin prescription is planned to avert ketosis and hypoglycemia precipitated by the low kidney threshold of pregnancy. The dietary prescription, therefore, is 30 calories per kilogram of increasing body weight: protein 2 gm. per kilogram of body weight, carbohydrate 180 to 250 gm. daily, and fat to complete the caloric prescription. The plan for insulin treatment is a prebreakfast dose of protamine zinc insulin plus crystalline insulin, usually followed by crystalline insulin before lunch and supper.

Estrogen and progesterone are prescribed to correct the hormonal imbalance. The sodium intake is restricted, protein increased, and ammonium chloride given to help overcome disturbed water balance.

By choice, both stilbestrol and proluton are prescribed as intramuscular injections, according to the daily dose schedule in Table 1. If oral stilbestrol is used, Table 2 is followed.

TABLE 1

Week of Pregnancy	Stilbestrol and Proluton Mg.
20	5 and 5
20-23	10 and 10
24-27	15 and 15
28-31	25 and 25
32 plus	50 and 50

TABLE 2

Week of Pregnancy	Stilbestrol Mg.
16	25
16-20	50
20-24	75
24-28	100
28-32	150
32	200

Therapy may be regulated by the clinical course as well as by laboratory tests. Thus, an abnormal gain in weight and signs of hydramnion and of toxemia indicate need for an increase in dosage. With such changes we advance to the next four-week period of our schedule. The assays for chorionic gonadotropin influence the prescription for stilbestrol, those for pregnandiol the dose of proluton.

DELIVERY

The plan for delivery is influenced by our fundamental concept of a diabetic pregnancy, namely, that of rapid maturity and rapid aging which terminate in a premature delivery or an intrauterine fetal death. To anticipate the latter, early timing for delivery is followed. By the method of trial and error, the beginning of the thirty-eighth week is elected.

If the cervix is not effaced, cesarean section is done under spinal anesthesia without medication. An infusion of 1,000 cc. of glucose is administered as a 10% solution preoperatively and repeated late in the afternoon of the day of operation. When long-acting insulins are used, the last dose is given twenty-four hours prior to surgery.

The immediate postnatal care of the infant includes [1] postural drainage, [2] aspiration of upper air passages at birth and every two hours for twelve hours, [3] aspiration of stomach at birth and every three hours for the first twelve hours, [4] oxygen incubation for five days, and [5] dehydration for two days by postponement of oral or parenteral fluid.

In the absence of advanced vascular disease, the expected fetal survival in patients so managed is 90%. The incidence of markedly premature deliveries is 3% and of preeclampsia 5%. But only when the genetic and vascular problems of diabetes are solved will the diabetic obstetric experience be equal to the best in nondiabetic obstetric experience.

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*Shipley, E. R. and Dodd, M. C.: Surg., Gynec. & Obst. 84:366. 1947. • McGivney, J.: South. M. J. 41:401. 1948

Arteriosclerosis in Diabetes

HENRY DOLGER, M.D.*
Mount Sinai Hospital, New York City

Prepared for Modern Medicine

THE subject of arteriosclerosis in diabetes mellitus all too often has been limited to consideration of late complications such as gangrene and peripheral vascular and coronary artery disease. The existence of earlier generalized lesions is now becoming generally recognized. To detect them, the physician must broaden the scope of his examination of diabetic patients, searching persistently for evidences of incipient vascular damage.

The usual analysis of urine and blood for glucose and the traditional catechism regarding the diet are inadequate for a complete evaluation of the patient's condition. Careful scrutiny of the fundi for early retinal hemorrhages, examination of the urine for albumin and casts, and determination of the blood pressure on each and every visit with the diabetic patient will reveal an astonishing incidence of abnormalities. Thereby will be disclosed a common paradox wherein the so-called mild diabetic patient may manifest severe complications and, conversely, the so-called severe diabetic patient may at times display mild vascular complications.

A common misconception concerning premature arteriosclerosis in diabetes is to regard it as a natural development in the patient beyond middle age. However, chemotherapy and the general improvement in diabetic care and education have removed the catastrophes of infection and coma from the forefront of diabetic morbidity and mortality. The young diabetic now lives long enough to develop accelerated vascular damage before middle age and, because the complication is so unexpected, it is shocking.

Reports from all over the world reveal but a handful of diabetic children who during the present insulin era have survived twenty-five years of the disease without premature arteriosclerosis. In a group of 60 juvenile diabetic patients, the early lesions of retinopathy were noted in 100% within an average of thirteen years after onset of the disease. At the same time almost 50% already had mild hypertension and albuminuria—associated phenomena of vascular degeneration. In some of the patients one or all three of the lesions were detected nine years after onset of diabetes, in others they appeared within twenty-two years.

The course of degeneration varied widely, with no apparent relation to the amount of insulin required or to the ability to control glycosuria. Rap-

* Adjunct Physician for Metabolic Diseases, The Mount Sinai Hospital; Instructor in Medicine, Columbia University, New York City.

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idly progressive lesions were noted in some young patients whose glycosuria was marked and almost constant, as well as in some with whom glycosuria was rare and mild.

Inexplicably, several juvenile diabetics, notorious offenders with respect to adherence to diet and displaying persistent glycosuria, presented only minimal lesions of vascular damage by the twenty-fifth year. Their fate was no worse than that of the youngsters whose observance of diet and management with insulin afforded no difficulty in preventing glycosuria. Some factor as yet unknown must explain the inherent resistance of the vascular systems of these two differently treated groups of young patients whose lesions progressed relatively slowly.

That hyperglycemia and glycosuria cannot be the determining factors in the development of diabetic degeneration must be conceded further in view of two unrelated phenomena. First, there are the verified instances of typical diabetic retinopathy in patients whose fasting blood sugar levels and even glucose tolerance tests reveal no abnormalities. Second, there are the now generally accepted observations that the birth of an excessively large infant (over 5 kg.) may portend the onset of diabetes in the near future for the mother. In both instances, the pathogenesis of diabetes, or of a metabolic disorder related to it, would seem to be operative long before hyperglycemia and glycosuria develop.

Not infrequently, patients are treated for so-called vascular complications long before the underlying diabetes is discovered. In not a few instances the possibility of glycosuria is first suggested by the ophthalmologist or neurologist, or even by the optometrist or podiatrist! It is noteworthy that the most advanced arteriosclerotic lesions are often seen in patients with so-called mild diabetes. The majority of the cases reported of the Kimmelstiel-Wilson syndrome or capillary glomerulosclerosis have been in just such mild diabetics.

The incidence of premature arteriosclerosis in the young and middle-aged adult diabetic as measured by retinopathy, hypertension, and albuminuria appears to be identical with that in the juvenile diabetic. Here, too, the earliest retinal lesions were noted about thirteen years after onset of diabetes and associated with hypertension and albuminuria in about 50% of patients.

An apparent acceleration of the lesions was typical of the mild cases in this older age group in whom glycosuria could be controlled by dietary regulation alone. Because of the insidious, undramatic onset of diabetes in middle age, duration of the disease cannot be determined accurately. But for all patients, both young children and adults, the only commonly accepted factor in the evolution of premature vascular damage is time—arteriosclerosis generally parallels the duration of the diabetic condition!

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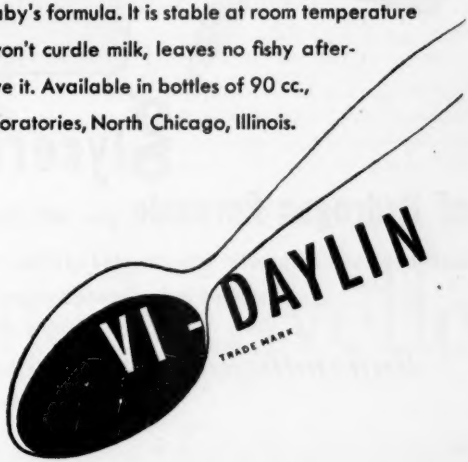

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A word about the commonly accepted index of arteriosclerosis, roentgen evidence of arterial calcification in the lower extremities. Inasmuch as nondiabetic individuals present positive findings in increasing numbers after middle age, this technic serves no discriminating function in the study of arteriosclerosis in the middle-aged or elderly diabetic patient. Its use in the juvenile diabetic too is unsatisfactory for this purpose, since only 11% of such a group of young patients had positive roentgen evidence of vessel calcification at a time when retinopathy had been noted in 100% and hypertension and albuminuria in almost 50%.

Since arteriosclerosis is accelerated in diabetes mellitus, regardless of age of onset, and since insulin, diet, and control of glycosuria have not significantly altered the course of the disease, the physician must search diligently for the earliest signs of such lesions in hope of finding the reversible stage in their development.

The past twelve-year experience with long-acting insulin preparations is too brief to verify the possibility that, with the nutritionally adequate diabetic diet of today, the improved metabolic balance obtained by these preparations for the diabetic patient may perhaps avert the dismal results of the past.

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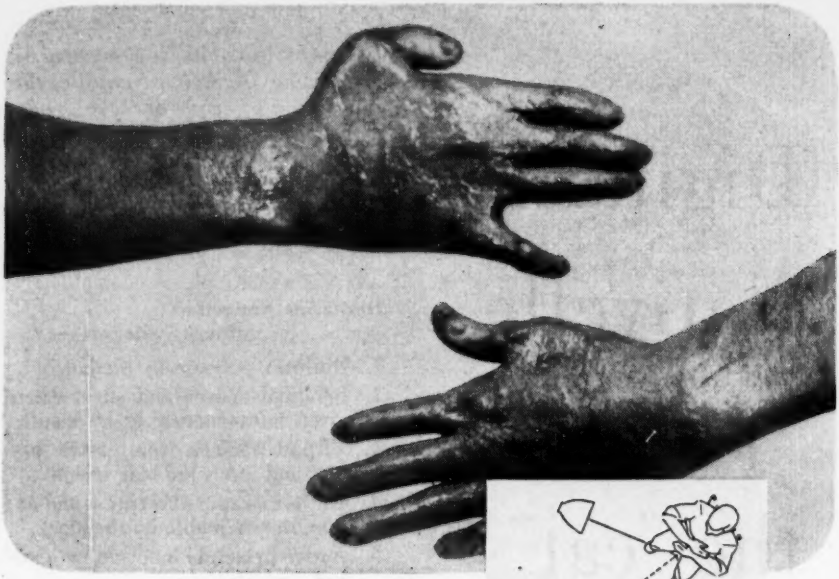

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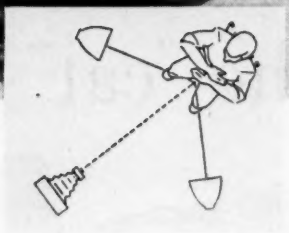

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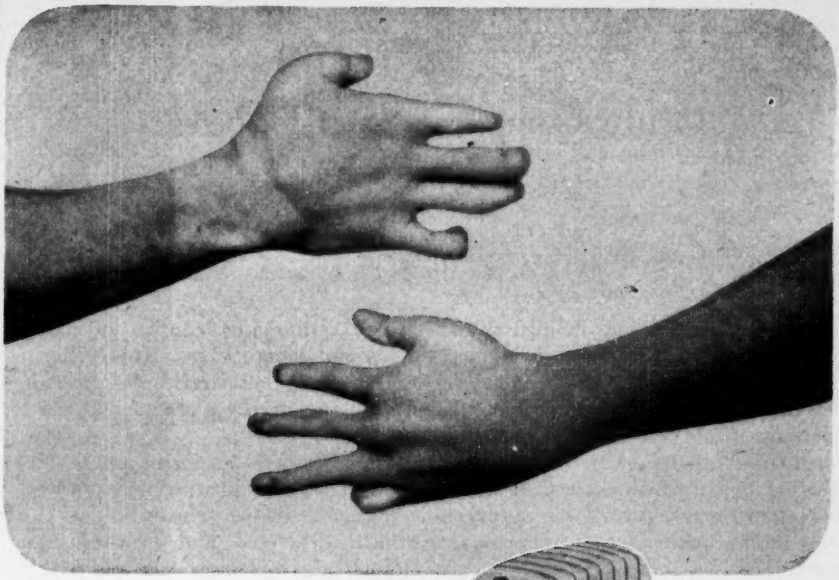

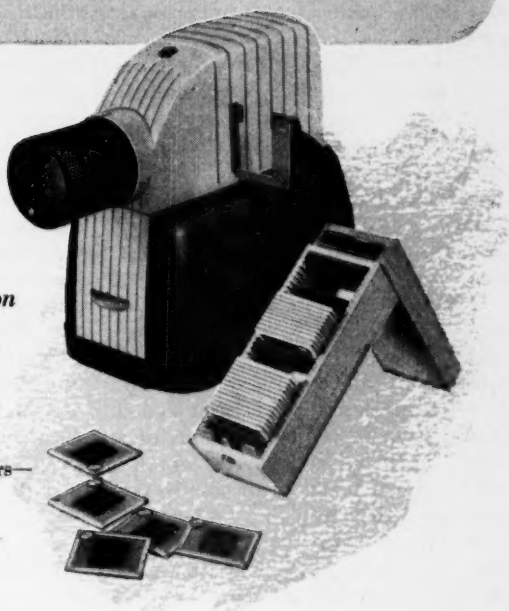

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Ocular Complications of Diabetes

J. Q. GRIFFITH, JR., M.D.*
Philadelphia

Prepared for Modern Medicine

PERSONS with diabetes mellitus show an increased incidence of cataract and retinopathy.

Cataract occurring in older diabetic patients is said not to differ from the ordinary senile lesion. In younger persons a characteristic snowflake type of cataract may occur, the lens appearing milky white from subcapsular opacities.

Cataract is believed to occur especially in persons who have maintained high blood sugar over a long period of time. A good diabetic regimen, therefore, may be valuable for prophylaxis in such patients, but once the cataract occurs the only treatment is surgery, undertaken at the proper time.

Retinopathy bears a definite relation to the duration of the diabetes but is less clearly related to the adequacy of the diabetic regimen. Indeed, it has even been suggested that a good regimen, in so far as it consists of a diet high in fat and low in carbohydrate, may actually predispose to retinopathy. Certainly the tendency in recent years is to give somewhat less fat and more carbohydrate, even though a greater insulin dosage is needed. However, it may take five or ten years to learn whether such change in diet will lessen the incidence of retinopathy.

The retinopathy consists of exudates and hemorrhages. The hemorrhages are characteristically small, round, and deeply placed in the retina.

Flame-shaped hemorrhages occur less frequently than with hypertensive retinopathy. The veins are apt to appear distended and over-full, as though their outflow was partially blocked in some manner and, indeed, thrombosis of a branch of the retinal vein, or even of the vein itself, is not uncommon.

Ballantyne[1] speaks of capillary dilatations which he calls microaneurysms and which may be confused with true hemorrhages. Such a lesion was constant for more than a year in fundal photographs loaned us by Donegan and Thomas.[2] Diabetic retinopathy may result in blindness from the continued destruction of retinal tissue by repeated hemorrhages and interference with blood supply, from hemorrhages into the vitreous, or from retinal detachment.

Once retinopathy has occurred, the vessels are already damaged, perhaps irrevocably, and probably the most that can be expected is arrest, not cure, of the condition. The treatment of diabetic retinopathy, therefore, consists of therapy for the underlying condition.

* John C. Griffith Jr. Research Foundation, Philadelphia.

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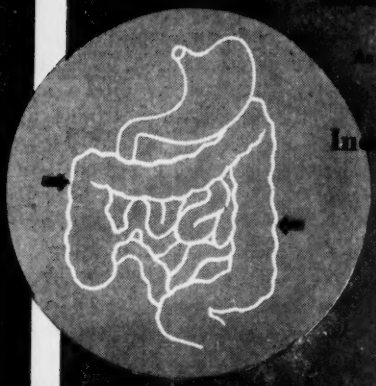

Schering

CAPILLARY FRAGILITY

In recent years, studies of change in capillary fragility and permeability have been related to various pathologic states. Certain substances have been found useful in modifying capillary fragility and permeability, and an extensive literature has grown up dealing with the so-called vitamin P type of substances.

Couch[3] has recently publish d a bibliography of 379 references covering the field. To summarize briefly:

► The early work in increased capillary fragility was related to clinical and experimental scurvy, which was treated by natural substances, such as fruit juices, containing vitamin C. Since vitamin C, or ascorbic acid, has become available in pure form, its relation to increased capillary fragility in scurvy is much less clear. While the evidence is sometimes conflicting, the bulk of the published papers suggest that increased capillary fragility is not a necessary part of scurvy and is not corrected by pure vitamin C.

► A series of more or less crude extracts from various plant sources have been prepared that are capable of correcting increased capillary fragility. To this group of substances the term vitamin P has been applied, although the justification of the classification as a vitamin is not certain. However, in the guinea pig or human subject on a scorbutigenic diet given pure vitamin C to prevent scurvy, the increase in capillary fragility can be corrected by a vitamin P preparation. According to this concept, classical scurvy is the result of the lack of vitamins C and P.

► Whatever the status of the vitamin P preparations as vitamins, they do contain physiologically active substances. Search for these substances in pure form continues and has included studies of rutin, hesperidin, and d-catechol.

Rutin, a glucoside derived from flavonol, $C_{27}H_{30}O_{16}\cdot 3H_2O$, was introduced into medicine by Griffith, Couch, and Lindauer[4] in 1943. Its use was first described in hypertensive subjects showing change in capillary fragility or permeability.[5] Later rutin was given to persons having retinal hemorrhage, with or without hypertension.[6]

The following test for capillary fragility, modified from Gothlin's method,[7] is recommended:

1] Mark off a circular area, 6 cm. in diameter, in each antecubital area. Mark off all blemishes and other signs in this area that might later be confused with petechiae.

2] Place a standard blood pressure cuff around each upper arm and maintain in each a pressure of 35 mm. of mercury for fifteen minutes. Lower the pressure. Count and mark all petechiae within the two circular areas, using a good light and a magnifying lens of 5 D or its equivalent.

3] One hour or more later, repeat, using a cuff pressure of 50 mm. of mercury. The petechial index is calculated as follows: To the number of petechiae occurring at 35 mm. of mercury multiplied by 2 add the additional number occurring at 50 mm.

The second pressure cuff application can be omitted:

► If the number of petechiae after the first application is 2 or less.

► If the number of petechiae after the first application is 6 or more.

► If the test is a repetition and

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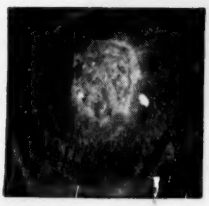

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may be compared with the corresponding stage or an earlier test.

The condition may be considered normal if the first petechia count is 2 or less, and in some cases even with a count of 3. A count of 6 or more is definite evidence of abnormality. The second application of the cuff may sometimes be necessary with a count of 3 and should always be made if the number of petechiae after the first test is 4 or 5.

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The test for increased capillary permeability depends upon measurement of cutaneous lymphatic flow by the dye method of McMaster.[8] The dye used is patent blue.

A minute amount of dye, usually about 0.04 cc., just enough to show beginning streamer formation, is injected intracutaneously. When the formation appears, injection is stopped. Since spread during the first minute is due to the force of the injection, the blue area is traced on cellophane one minute after injection and again after fifteen minutes.

If spread from the one-minute to the fifteen-minute tracing is more than ¾ in., measured in a straight line, flow is considered increased. In the absence of renal or pituitary disease, increased cutaneous lymphatic flow is considered to indicate increased capillary permeability.

(Continued on page 94)

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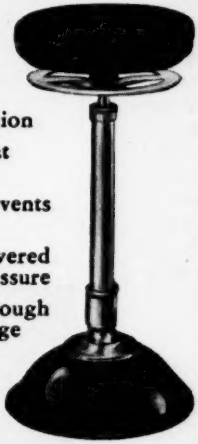

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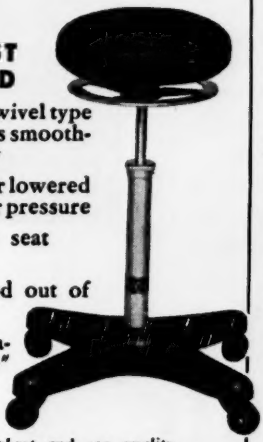

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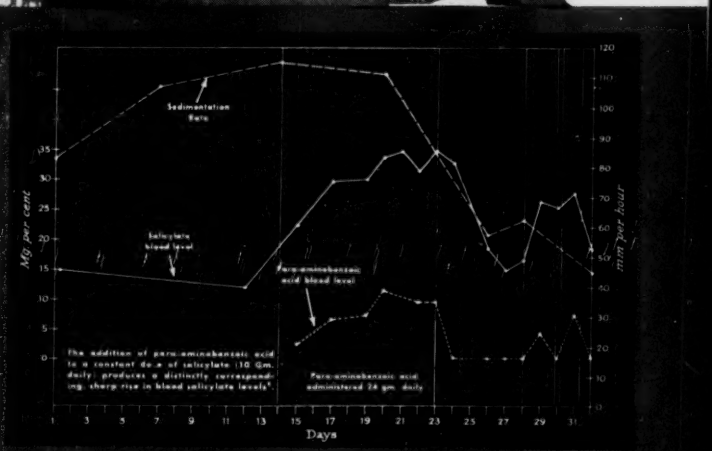
Sedimentation Rate
Salicylate blood level
Para-aminobenzoic acid blood level
Mg. per cent
mm. per hour
Days
The addition of para-aminobenzoic acid to a constant dose of salicylate (10 Gm. daily) produces a distinctly corresponding sharp rise in blood salicylate levels.
Para-aminobenzoic acid administered 24 gm. daily.

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RUTIN THERAPY

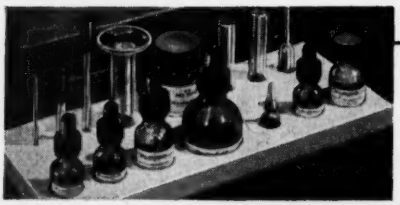
A patient with increase in capillary fragility or permeability or both is said to have a capillary fault. In the hypertensive group of 1,540 subjects studied by the author, 18% had a capillary fault. Retinal hemorrhage prior to first visit was reported in 9.3% of those with capillary fault, and in 2.6% of the others. During an average sixteen-month period after initial visit, retinal hemorrhage occurred in 0.8% of persons without capillary fault, in 0.4% of persons with an initial fault which had become normal after rutin therapy, and in 3.8% of those with initial capillary fault who had been either untreated or treated inadequately with whom tests were persistently abnormal.

Thus, in the hypertensive group, it seemed fairly probable that rutin, given to the point at which abnormal tests became normal, could prevent retinal hemorrhage with certain patients.

When rutin was given to patients who had suffered a previous hemorrhage,[8] results were less striking, but repeated hemorrhage occurred in 5.7% of persons whose capillary fault had been corrected by therapy, and in 38.3% of those whose fault persisted.

Certain patients in our series were diabetic and we noted that, in general, they did not get along as well as the nondiabetics, but more adequate studies of diabetic subjects have been made by Rodriguez and Root,[9] Donegan and Thomas,[2] and Beard-

20-minute diagnosis of pregnancy



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wood, Roberts, and Trueman.[10] From these investigations it appears:

► About 40% of diabetics show capillary fault.

► Of those with retinopathy, nearly 100% show capillary fault.

► Under adequate rutin (or hesperidin plus vitamin C) therapy, capillary fault can be corrected or at least improved.

► Once the retinopathy is established in the diabetic, it does not regress when capillary fault is corrected, as it does in the hypertensive.

► However, when capillary fault is corrected such treated cases tend not to progress or at least vision appears to be preserved, as a rule. This apparent discrepancy may be explained, in part at least, by the occurrence in the retina of dilated capillaries which may be confused with hemorrhages; hence a static process may be considered active.

RUTIN DOSAGE

The dosage of rutin and the duration of treatment must be considered in evaluating results. Certain reports have appeared in which one or both have been inadequate, and negative results secured. A reasonable regimen would appear to be as follows:

For the diabetic *without* retinopathy, tests for capillary fault should be carried out at regular intervals, perhaps every three months. If one or both become abnormal, rutin should be given, 20 mg. three times daily, and abnormal test or tests re-

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peated in six weeks. Every time a test is repeated and found to be abnormal, rutin dosage is increased. Since rutin is not toxic, final dose may be high, in our series as high as 600 mg. per day.

When repeated tests are normal, dosage is merely maintained and the interval between tests may be lengthened to three months. This is the group which offers the most promising field for the future.

For the diabetic *with* retinopathy, the regimen is the same except that the initial dose should be higher, 60 mg. three times daily. Prognosis is less favorable. However, Joslin[11] states, "It is the impression of Dr. Priscilla White, who has followed the progress of our cases of retinitis proliferans, especially in young diabetic persons, that there has been much less rapid progress of these lesions since the introduction of rutin."

The administration of rutin without preliminary testing is not recommended, but if it is so used, chance for benefit is greater with the higher doses, such as 200 to 300 mg. per day.

OTHER CONSIDERATIONS

A search for other causes of hemorrhage should be made or at least considered. Regardless of theoretic considerations, if there is any possible question of lack of vitamin C, it should be supplied in excess.

Prothrombin time should be measured. If prolonged, vitamin K should be given until the time is corrected.[7]

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If it is shortened, McLean and Brambel[12] give dicumarol with the rutin to maintain prothrombin activity at 50 to 60% of normal for six months or more. Schneider, Lewis, and McCullagh[13] report a small series with low albumin and corresponding rise in beta globulin. Retinopathy in these patients, all diabetics, was benefited by high-protein feeding. The improvement was accompanied by a rise in albumin and a corresponding drop in β-globulin.

Whatever the final verdict of time on the methods of therapy outlined above, the principles on which they are based appear to be sound.

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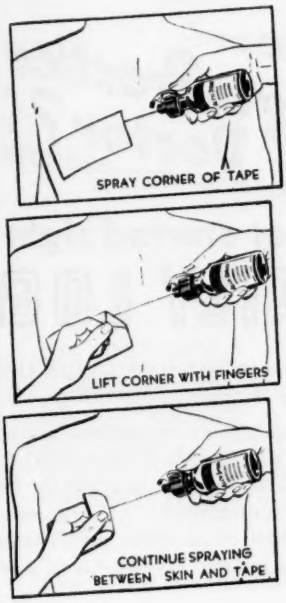

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CLINICAL SOCIETIES

The main function of the Clinical Society is the promotion of education in the science of metabolism within the ranks of the medical profession. To accomplish this end, it fosters formal open lectures and lecture courses, but in addition does its own "family washing" behind closed doors by means of medical conferences. In these gatherings, the title "Doctor" is cast aside and members Tom, Bill, and Jim let down their hair, exchange experiences, argue out and crystallize scientific methods.

In its own community the Clinical Society establishes minimal standards of facilities and treatment to be met by local diabetes clinics and certifies only those which live up to these standards. Membership in the Clinical Societies is graded—Fellowship is bestowed on recognized specialists in the field. Such recognition depends not on the superficialities of the physician's ability to pass an examination, but on his actual standing in the medical community as gauged by his professional peers.

LAY SOCIETIES

On the other hand, the Lay Society with its lay officers is composed not alone of diabetics but of any lay persons who for one reason or another, often because of diabetic friends or relatives, are interested in diabetes. In this way membership in no wise identifies an individual as a diabetic

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Boasting its autonomous nature independent of medical rule, the Lay Society holds open public forums in which specialists from other cities are invited to address the laymen and answer questions propounded by the lay listeners. Through the prestige and influence of its sister society, the Clinical Society, the Lay Society is able to commandeer for its educational ventures the outstanding medical talent of the entire United States and Canada.

The local physicians sit most unobtrusively in the audience and never address the laymen of their own community. They interestedly watch their confreres from other cities sweat through the occasion, realizing that they in turn will be reciprocating for some Lay Society in a community other than their own. Two physicians of the Clinical Society are invited by the laymen to give counsel behind the scenes as to projects and programs.

Both professional and lay societies join hands in such major projects as the financing of summer camps for diabetic children and in matters involving the social welfare of the diabetic, including life insurance and employment problems.

The chief value of the entire setup is the fact that the layman is made to feel that he has a personal duty in the fight against diabetes, that he is a free agent and should assume his place and obligation on an equal footing with his physician. He usually grasps the challenge with avidity, for it is *his* own disease in which interest is being courted.

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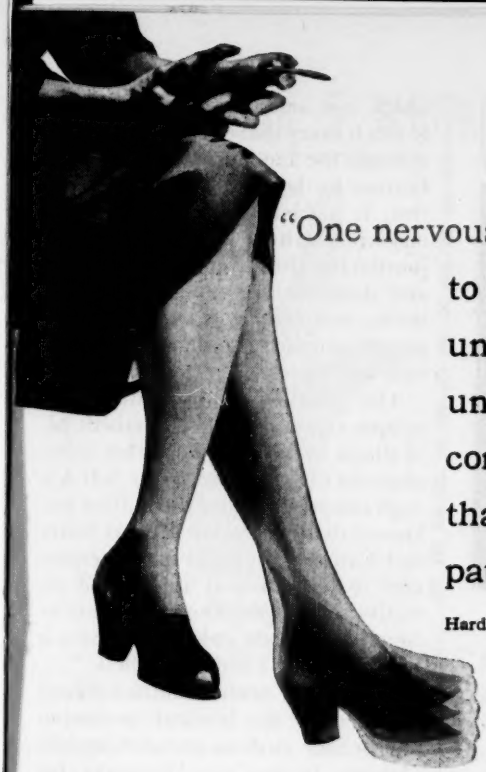

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which, it is anticipated, will ultimately reach every diabetic in the country through the Lay Societies. This publication for laymen, the *A.D.A. Forecast*, is a bimonthly, digest-type of magazine, written by specialists. The journal has already entered the homes and daily lives of thousands of diabetics, and articles from it have appeared abroad, translated into Finnish and Dutch.

The guiding genius behind this unique creation is Mrs. Elizabeth M. Mullann of Philadelphia. Her influence was demonstrated in the A.D.A.'s 1948 campaign to find the million unknown diabetics in the United States and Canada. Response was phenomenal to her editorial appeal and directive: "Let every known diabetic in America find one unknown." "Save a life by finding a hidden diabetic."

Laymen in both countries joined hands with the medical profession and became modern crusaders against diabetes. In the New York area, for example, four detection centers established by the New York Diabetes Association were manned by some sixty eager diabetic laymen who volunteered to serve as receptionists, secretaries, and technicians at the side of the doctors who officiated as members of the Clinical Society.

With the religious zeal engendered by this type of physical setup, diabetes must inevitably, in the not too distant future, join the ranks of the already conquered enemies of mankind, diphtheria and tuberculosis.

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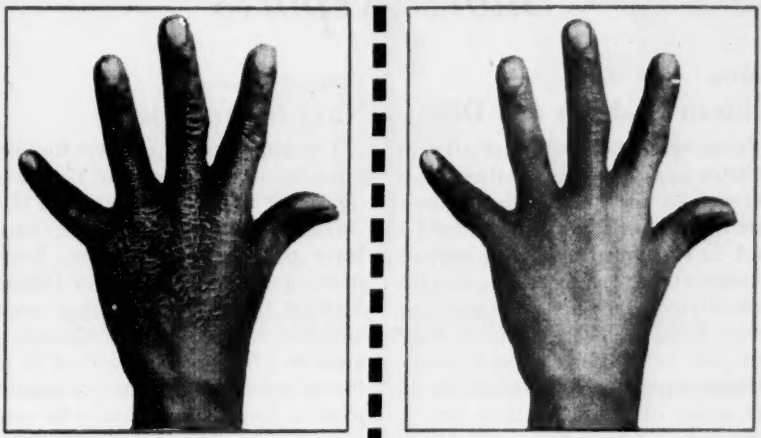

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M. J. Australia 1:121-126, 1949.

EDUCATION

Navy Internships

The appointment of three hundred candidates to the grade of lieutenant (jg) in the Medical Corps of the U. S. Naval Reserve is planned under a new Navy procurement program. Intern training will be pursued in Civilian Medical Centers during 1950 under auspices of the Navy. Candidates must be graduates or enrolled in the fourth year class of approved medical schools. Applications should be made through the Office of Naval Officer Procurement nearest the candidate's place of residence.

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PUBLICATIONS

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Much question-answering by the doctor may be eliminated by a book that has just been published under the title *Having Your Baby*. The volume includes a *Modern Medicine* special article, "Alleviation of Anxiety during Pregnancy," by Dr. Leonard Biskind of Cleveland, and the author's companion paper "Modern Prenatal Care." Thousands of reprints of the special article were requested by physicians for their obstetric patients. The book published in attractive format by the Western Journal of Surgery, Portland, Ore., should prove equally popular.

EDUCATION

Teaching by Television

Physicians attending the AMA convention in Atlantic City in June will see surgical operations and diagnostic procedures televised in natural color by the medical staffs of the University of Pennsylvania and Atlantic City Hospital. Pictures will be shown continuously for four days in Convention Hall as a pioneer demonstration of the possibilities of television for teaching medicine and surgery.

J. A. Am. M. Coll. 24:123, 1949.

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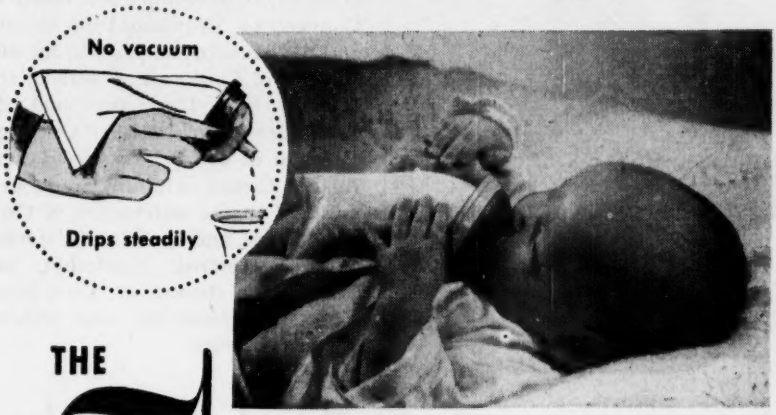

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sion of cancer programs, the National Cancer Institute has announced the approval of special control grants totaling $170,205 and teaching grants of $261,416. Control grants have been made to the departments of health of Connecticut, Maryland, Pennsylvania, and the Territory of Hawaii; the universities of California, Colorado, and Illinois; Yale University; and the Radiotherapy Institute and Oncologic Hospital of the Puerto Rico League Against Cancer. Training grants were awarded to the universities of Georgia, Illinois, and Southern California; Boston, Howard, Vanderbilt, and Washington universities; Long Island College of Medicine; and Meharry Medical College.

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The Insulins in Diabetes

References for the paper on pages 64-67 by Franklin B. Peck, M.D.

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Sign Language

A foreign-looking man came in to see the doctor who was out. I asked the man what he wanted to see the doctor about.

He waved his hands excitedly and kept repeating, "I want peace! I want peace! I want peace!"

When he realized I didn't understand he started to unzipper his trousers to make his meaning clear. I left hurriedly to locate the doctor.—T.E.

"I took a first-aid course and analyzed my own symptoms, Doc, will you sign the prescription?"

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A woman and a small boy were sitting alone on a bench in the almost deserted clinic. They stopped me as I walked by. The woman asked, "Is this where we sit for worms?"—D.S.A.

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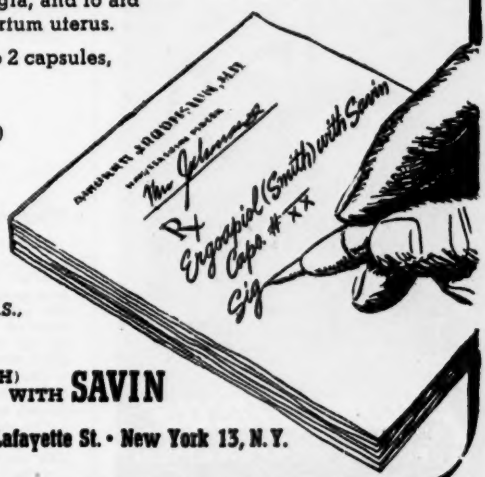

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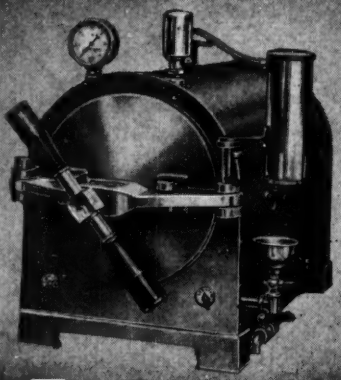

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"Doctor," said the young widow, "can you give me something to make my periods regular? Innocent or guilty, I worry if I don't menstruate every twenty-eight days."
—D.B.

Dr. Jekyll, Mr. Hyde

One day as I was interviewing a farmer concerning his disabilities I asked, "Do your hemorrhoids give you any trouble?"

"No, sir, not a bit."

At the close of the interview I asked, "Is anything else giving you any trouble that we haven't mentioned?"

The patient leaned over and whispered, "Yes, sir, my piles."—H.E.C.

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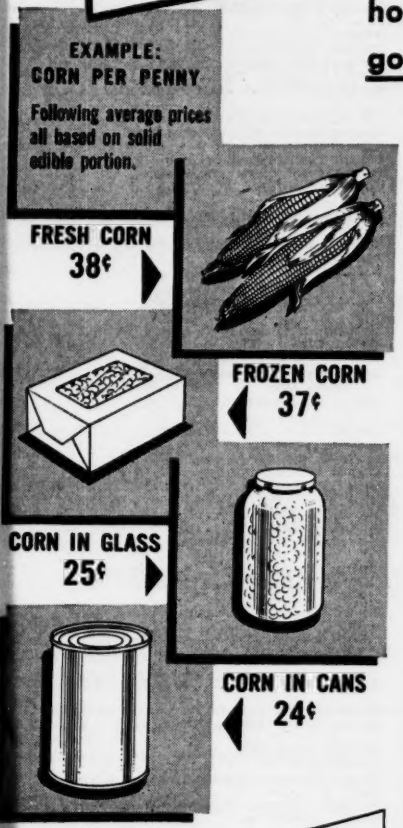

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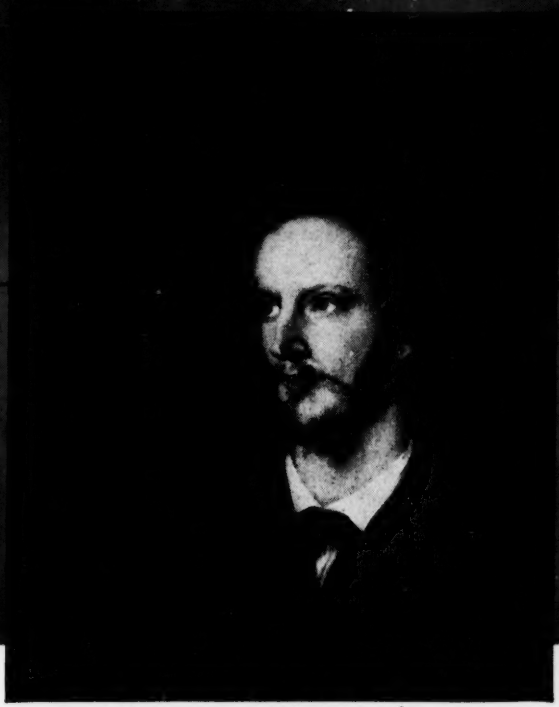

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1. Grimson, Marzoni, Reardon and Hendrix: Ann. of Surg., 127: 5, May, 1948.
2. Reich, N. E.: Med. Times, Jan., 1949.

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